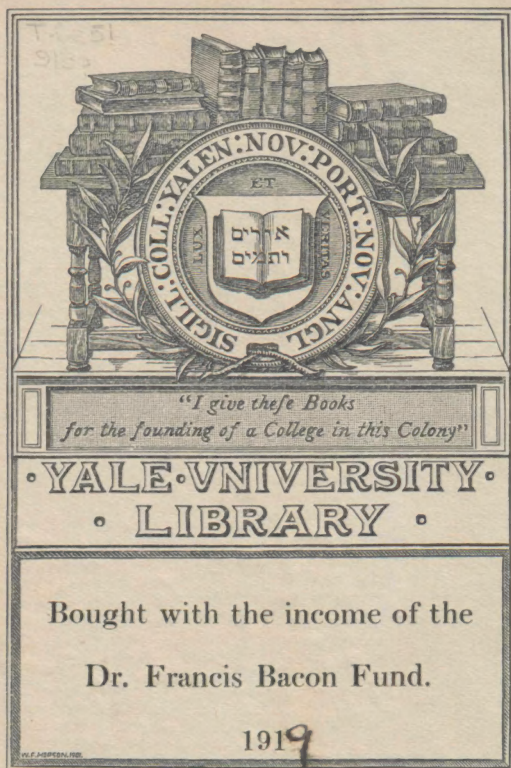


THE INFLUENCE OF SUNLIGHT
IN THE PRODUCTION OF
CANCER OF THE SKIN

C. NORMAN PAUL



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THE INFLUENCE OF SUNLIGHT IN THE PRODUCTION OF CANCER OF THE SKIN

BY

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TO
THE MEMORY OF MY BROTHER
ALBERT RAYMOND PAUL
AUSTRALIAN IMPERIAL EXPEDITIONARY FORCES
FRANCE

PREFACE

THIS small book demonstrating some common cancerous and precancerous conditions of the skin, in which sunlight plays an important part in their production, is written with the object of bridging a gap in English text-books on dermatology, where these diseases are usually passed over in a cursory manner. The Radium Department of the Sydney Hospital has afforded me exceptional opportunity in the study of these diseases.

I have to acknowledge my indebtedness to the writings of various authors, and particularly to those of Dr. H. G. Adamson.

235, MACQUARIE STREET, SYDNEY, AUSTRALIA,
July 1917.

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THE INFLUENCE OF SUNLIGHT IN THE PRODUCTION OF CANCER OF THE SKIN

INTRODUCTION

THE most important cutaneous lesions in this country, viewed from the standpoint of their prevalency and destructive effects, are those malignant diseases which are especially prone to occur on exposed parts, foremost amongst which are rodent ulcer and epithelioma. Factors which play a significant rôle in their causation and prevention are the actinic rays of light, and the pigmentation of the skin. If we follow the geographical distribution of the coloured races, in whom the pigmentation of the skin is most pronounced, we find that they occupy tropical and sub-tropical countries, in which, although the effects of sunlight are most severe and long-continued, its action upon them is minimal. This is verified by the opinion of authorities on tropical diseases, and Castellani states that these diseases are rare in the natives of Ceylon.

It will, therefore, be seen that the melanin, the pigment of the skin, stands as a sentinel, guarding the underlying tissues from the baneful effects of sunlight. In the white races this pigment is confined for the most part to the periphery of the cells of the basal layer of the epidermis and to the lowermost stratum of the prickle-cell layer,

whilst the pigment granules may also be found in the interepithelial lymphatic spaces, and in the fusiform connective tissue cells of the papillary body. In the dark races, Macleod states that pigment is to be found as high up as the transitional layers of the epidermis, as well as in some of the connective tissue cells of the superficial portions of the corium. But the white races have the power of reacting to the external stimulus of light with an increase in the amount of pigmentation. This phenomenon is to be seen in those who are suddenly exposed to strong sunlight, when unaccustomed to it. Almost immediately the subjacent tissues become swollen and painful, and the skin assumes a bright red colour, followed by desquamation (*erythema solare*). Later, a more permanent brownish pigmentation is produced, which has the property of excluding the actinic or the ultra-violet rays of the spectrum. The above-described results are to be attributed to these rays, and not to the effects of heat. The common occurrence of these cancerous and precancerous diseases of the skin in Australia is to be regarded as one of the penalties to be paid for inhabiting a country normally destined to be occupied by a coloured race.

With the introduction of radium, the successful treatment of these conditions has advanced with giant strides, and it has now become an unusual incident to see the large ulcerations, the so-called terebrant type of rodent ulcer, the heritage of the surgeon.

XERODERMA PIGMENTOSUM

DEFINITION

Xeroderma pigmentosum, first described by Kaposi in 1870, is a disease in which there is a congenital defect of the skin. It reacts to the external stimulus of light, with the production of freckle-like pigmentation, telangiectases, atrophic spots, warty growths, indolent malignant ulcers, and epitheliomata.

ETIOLOGY

Although there is a congenital predisposition, the influence of the actinic rays of light must be regarded as the exciting cause. Statistics show that males and females are equally affected. It may occur in more than one member of a family, and when this happens the disease may even be limited to a particular sex. In the majority of cases the onset of the malady is either during the first or the second year of life.

PATHOLOGY

The pathological changes first appear in the papillary body and the epidermis, and exhibit degenerative changes of an atrophic nature. The following is the interpretation of the disease placed upon it by Unna:—

1. *Freckle-like pigmentation* is due to an excess of melanin, extending to the horny layer and to the corium.
2. *Telangiectases*.—Their development is related to

the white spots. From the diffuse capillary hyperæmia, by disappearance of many capillaries, there arises a limited collateral hyperæmia, with resultant ectases of the venous capillaries. No new formation of vessels has been recognised in connection with the red lines and points.

3. White spots are due to hyperplasia of the connective tissue fibres. Only the circulatory system undergoes atrophy, and in point of fact the epidermis and the collagenous tissue are hypertrophic.

4. *Malignant tumours*.—Four examined were all true carcinomata and unpigmented. They developed rapidly from the surface, and were of the fungating form. Crocker states that in one of his cases the tumours were distinctly papillomatous, and not malignant for many years. Melanocarcinomata have occurred, and Kreibich has described cases belonging to the medullary cancer type.

SYMPTOMS

The early manifestations are to be observed during the first or second year of life, but most frequently in the first summer after birth. Those cases that occasionally appear in adults, I prefer to classify under *dermatitis solaris chronica*. The first stage may be recognised by: (1) An erythematous condition of the skin, in which there is a dry, scaly roughness. (2) Later, pigmentation, in the form of freckles, appears, affecting the face, neck, shoulders, forearms and hands. Occasionally the portion of the legs below the knees may be affected. These are the evident sites of exposure in children. The pigmented spots increase in number, and show little tendency to disappear in the winter. They vary in size, and are of a yellowish brown colour. (3) White atrophic patches may be seen

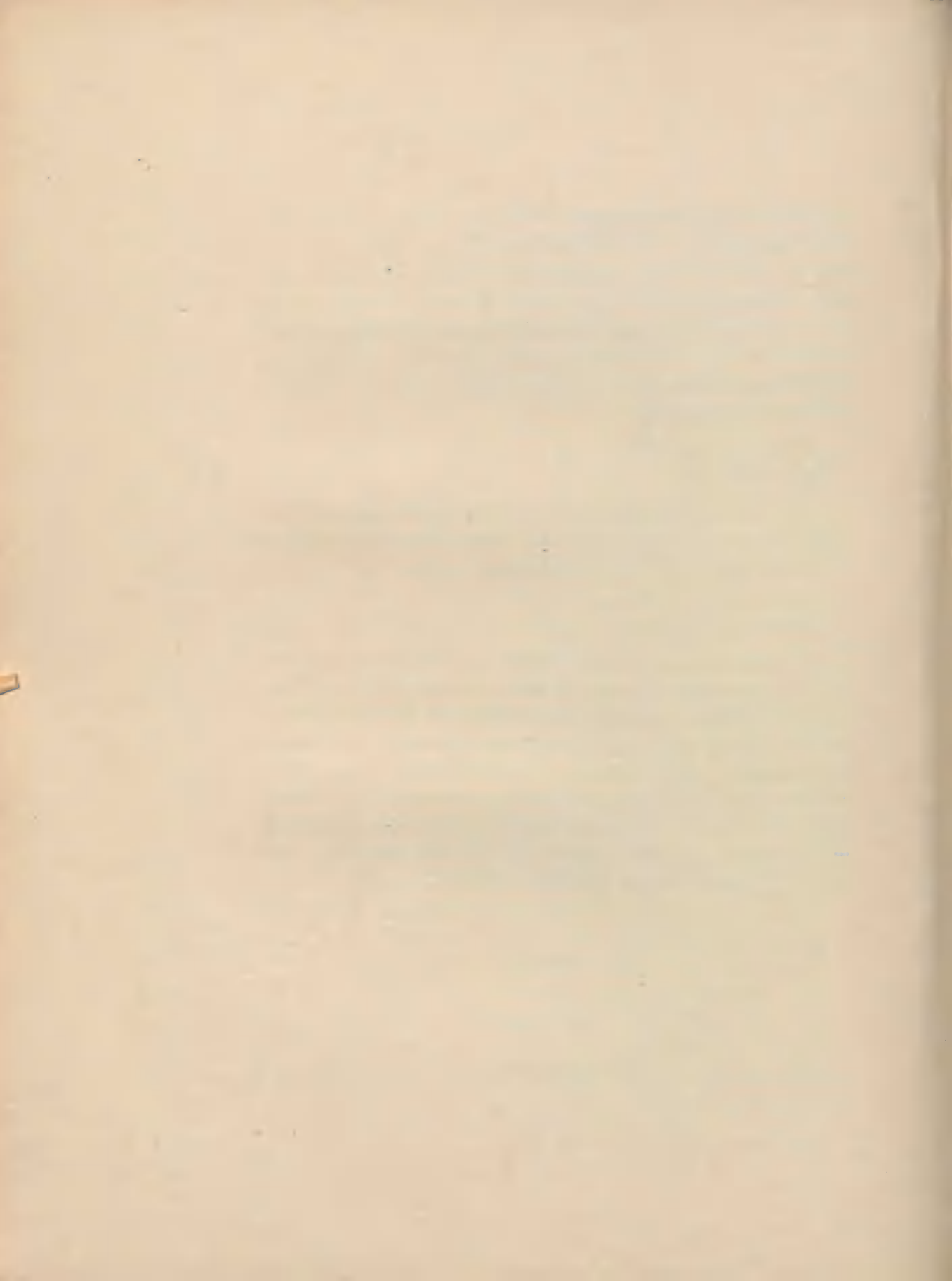
on the face, and in the region of the eye may coalesce, and cause extropion. (4) Dilated vessels, or telangiectases, may be in evidence amongst the freckles, and upon the white atrophic spots. (5) Warty growths develop later, and occasionally carcinomata and superficial ulcers of an indolent nature owe their origin to these. The disease may run a rapid course, or there may be periods of intermission and quiescence.

PROGNOSIS

Patients usually die at an early age, from the development of carcinomatous growths, which sometimes produce metastases in the internal organs.

TREATMENT

Treatment is mainly palliative. Protection from sunlight is essential, and can be aided by the wearing of red or brown veils, or the application of similarly coloured ointments. When the warty growths develop, radium or Röntgen rays may be used. The same mode of treatment applies to the indolent superficial ulcers and the commencing malignant growths, or if necessary they may be excised. If these measures are adopted, the life of the patient can often be prolonged for a number of years.



RODENT ULCER

DEFINITION

A chronic and local malignant disease of the skin, characterised by slowly progressive ulceration, preceded by a nodular stage. The disease was first described by Jacob of Dublin in 1827, and it was then that he drew attention to many of its essential features. Following his description, it has been the custom with English authorities to describe it as a separate disease. Although we occasionally meet with indefinite lesions, which it may be necessary to submit to pathological examination, the condition is usually sufficiently distinctive to enable us to make a clinical diagnosis, and to warrant us separating it from ordinary epithelioma of the skin.

ETIOLOGY

The disease is mainly confined to middle or advanced life, but cases manifest themselves in childhood, Sequeira having recorded one at the age of twelve. The following table gives a record of the last twenty-five cases of rodent ulcer seen by me in hospital practice in the past few months, having regard to the age and sex of the patient and the site of distribution.

Sex.	Age.	Site of Distribution.
Female	34	Left inner canthus
Male	56	Ear and nose
Female	67	Behind the right ear
Female	65	Left malar region

Sex.	Age.	Site of Distribution.
Male	59	Left lower lid
Male	72	Left nasofacial groove
Female	65	Left lower lid
Female	54	Nose, near inner canthus
Male	47	Right nasofacial fold
Female	65	Nose
Male	75	Right temple
Male	61	Inner canthus right eye
Male	80	Nose
Female	24	Forehead
Male	77	Lower lid
Male	78	Nose
Female	35	Malar region
Male	74	Nose and right inner canthus
Male	60	Left nasofacial fold
Male	55	Left side face
Male	71	Left inner canthus
Female	47	Right inner canthus
Male	47	Right parotid region
Female	53	Nasofacial fold
Male	37	Beneath left eye
Male	37	Malar region

These neoplasms may arise from (1) a nodule ; (2) a local injury ; (3) unpigmented or other moles ; (4) scars ; amongst the accompanying photographs will be observed cases demonstrating these various modes of origin ; (5) they have often been described as originating from keratoses, and Radcliffe Crocker saw a case which started from a yellow plaque.

PATHOLOGY

The vague doubt that existed years ago as to the site of origin of rodent ulcer remains even to-day. English authorities formerly considered it to be a disease *sui generis*, independent of epithelioma, and to arise from either the hair follicles, the sebaceous glands, or the sweat

glands. Many continental writers, who previously did not differentiate between rodent ulcer and epithelioma, have now come to regard the former as an epithelioma derived from the hair follicles. To the original continental view Unna was a prominent exception, for he differentiated rodent ulcer from ordinary epithelioma of the skin. American dermatologists for the most part adhere to the continental opinion, and describe them as slowly growing epitheliomata. To-day, however, there is an increasing tendency to follow the classification of Krompecher, and to designate them baso-cellular carcinomata, (that is, a carcinoma arising from the basal-cell layer of the epidermis, or the corresponding portion of the hair follicle) in contradistinction to the ordinary spino-cellular or prickle-cell carcinomata, on the ground that spines are absent in the former. Adami, in criticising this classification, states that it is based "on the mistaken ground that it shows no prickle cells or keratinization, it is derived wholly from the undifferentiated cells of the *rete Malpighii*. But this is so also for all epitheliomas. In the more highly developed forms the prickle cells present do not arise from pre-existing prickle cells, but also from the basal mother cells. It is the stage of undifferentiation or anaplasia, the capacity or incapacity to develop beyond a certain point that determines the form of the cells." Rodent ulcer was noted by Sir Benjamin Brodie to arise from moles.

A step further is taken in this direction by Adamson, who looks upon rodent ulcer as being a nævoid growth, derived from the basal-cell layer of the epidermis. In the *Lancet* of March 21st, 1914, p. 814, he states, "Rodent ulcer is a delayed attempt to form hair follicles and sebaceous glands, in which the effort does not extend beyond mere proliferation of those cells which at an early age would have

become differentiated into these special organs. It is an illustration of the Cohnheim theory of cell-rests."

Examination of sections of prickle-cell carcinomata discloses the fact that there is always a definite and extensive connecting link between the underlying growth and the epidermis; it is unnecessary to obtain serial sections to prove this fact, so that the origin of the growth from the surface epidermis is demonstrated beyond doubt. In rodent ulcer this evident connection of the growth with the surface epidermis is for the most part either somewhat indefinite or absent. In sections the hair follicles are often cut in such a way as to give the impression of a downgrowth from the epidermis, especially if they show proliferation at their junction with the epidermis. We may assume that if rodent ulcer took its origin frequently from the surface epidermis,—and we must admit that this does occasionally happen,—then the connection between it and the underlying growth would be more constant, as it is in epithelioma. It must, however, be conceded that the hair follicles are frequently the starting-point of rodent ulcer. The bur-nished appearance of the surface, and the telangiectases, may be entirely due to a sub-epidermal growth, or it may be simply due to a lateral proliferation of a growth that has arisen from the surface epidermis.

Histologically, rodent ulcer is composed of cylindrical processes of epithelium, which branch and spread laterally. The cells forming these processes are closely packed together, have large nuclei in comparison with the size of the cell, and are somewhat spindle-shaped, without prickles, and limited by a definite palisade layer. The fibrous tissue is condensed, and may become scirrhus, and emits trabeculæ between the epithelial masses. It is denser in the depth than at the margin of the growth, where there

are collections of plasma cells, and it offers a barrier to the spread of the disease, which partly accounts for the early growth being superficial and peculiarly hard, and for the vessels coursing over its border as they take the path of least resistance. Dubreuilh and Auché (*Annales de dermatologie et de syphiligraphie*, Aug.-Sept. 1901, p. 705) state that "the dura mater is very resistant to the invasion of the growth; in striped muscle a kind of interstitial myositis results, and the fibres become separated by the growth of fibrous tissue between them. In bone, the epithelial processes penetrate the Haversian canals, but are always separated by a thin layer of connective tissue. The nerves are destroyed by an interstitial sclerosis." In short, the fibrous tissue barrier plays an important part in the pathological picture of this type of growth. Another characteristic feature is the absence of any definite small cell infiltration such as is seen in ordinary epithelioma. In ordinary rodent ulcer the microscopical appearance of sections differ to a certain extent. The density of the fibrous tissue stroma seems to vary in proportion to the rate of progress of the growth, and the arrangement of the masses of cells to the surrounding stroma is also variable.

SYMPTOMS

Rodent ulcer is a disease which generally affects the face. Its site of predilection is the central horizontal third, that is, the area bounded inferiorly by a line drawn just beneath the nose to the lobule of the ear, and limited above by a line extending posteriorly from the superciliary ridges. The majority of the cases arise from the inner and outer canthi, and the nasofacial groove. Cases have occasionally been recorded which demonstrate that the

disease may affect any part of the body. Amongst others, Walker describes cases as occurring on the scalp, forearm, back of the hand, pubis, and vulva, whilst Sequeira reports rodent ulcer as occurring on the shoulder, beneath the scapula, in the mammary region, and on the back. Although it is generally single, it may at times be multiple, independent of the condition described under multiple rodent ulcer.

In considering the early manifestations of the disease one has often to be guided by the statements of the patient, who somewhat loosely describes the early lesion as a pimple or warty growth. Usually the first indication of the affection is a small nodule with a burnished appearance, having telangiectases over its surface. The growth may present itself thus for many months, or even years, before breaking down in the centre, and forming a scab, which is liable to be frequently removed in washing. The ulcer now gradually enlarges, the border becomes rolled or beaded, but it is not undermined or everted, and has dilated capillaries coursing over it. The beaded border may completely encircle the growth, or may be present at only a portion of the periphery. Sometimes there is an indifferent border to the ulcer and the rolled beaded edge is absent. Cases have been recorded where the early lesions have reached a large size before ulceration has taken place, and Bowlby reports a case which reached the size of a tangerine orange. It advances slowly and steadily, and takes years to reach an appreciable size. It tends to progress laterally rather than deeply, and differs from epithelioma in that the amount of new growth is small, even when there is extensive destruction of tissue. There is but slight discharge from the base of the ulcer. Every type of tissue which it invades is destroyed, and once

the subcutaneous fat is reached, its progress is rapid. Rodent ulcer may assume a superficial type, and form a shallow ulcer of even depth. These ulcers cicatrise, and spread simultaneously. In them the beaded border may be but imperceptibly raised, the ulceration, however, may affect a large area. I recently saw a case of this nature of many years' duration, extensively involving the right side of the face.

When the deeper tissues become invaded, and this is often a sequel to operations at which a sufficient margin of tissue has not, or could not be removed, the growth spreads more rapidly, for the protective fibrous tissue barrier has been broken down. These cases form what is known as the terebrant type of the disease. They are most frequently to be seen starting around the auricle, the nasal, buccal orifices, and the orbital cavity, and may lead to the entire destruction of these parts. Enucleation of the eye for rodent ulcer is not infrequently followed by extensive ulceration of the orbital cavity, as the growth soon involves the orbital fat, and sepsis aids in the destructive process.

Rodent ulcer may commence on the eyelid, usually the lower, adjacent to the conjunctival border. It is here conspicuous, for it often appears as a whitish spot on the conjunctival surface, and slowly increases in extent. On pressure it appears cartilaginous. This sign is frequently to be noted in all forms of rodent ulcer, and when it is pressed between the fingers there is often given the impression of a coin being felt between cloth. Slight pruritus may occur early, but pain as a rule is not complained of, except in the terebrant type of cases. The lymphatic glands throughout remain unaffected, and metastasis does not occur.

DIAGNOSIS

In considering the diagnosis of rodent ulcer attention must be paid to its usual site of distribution, as well as to its extremely slow rate of progress. The early rodent nodules are generally single and of a pearly appearance, and often show telangiectases coursing over them. When ulceration has taken place they are characterised by their pearly beaded border, which also frequently has fine vessels coursing over it. Occasionally this beaded border is absent. The discharge is scanty, at least until the deeper tissues become involved, but is more copious when sepsis is present. The lymphatic glands are very rarely involved.

DIFFERENTIAL DIAGNOSIS

(a) *Lupus erythematosus*.—In the sebaceous type of lupus erythematosus superficial ulceration may take place, and thus may resemble the superficial type of rodent ulcer. In both, the base of the ulcer is free of discharge and without granulations. In lupus erythematosus there is a persistent, dull red, inflammatory border, which is absent in rodent ulcer, and there may be some comedones. In rodent ulcer there is the slightly raised pearly border, which may be present at only one portion, indicating the active part of the growth.

(b) *Syphilis*.—Gummata may resemble rodent ulcer, but there is often a history of the ulcers being preceded by a hard inflammatory nodule, which breaks down with a punched-out, sloughing, and discharging base; moreover, they are often multiple. The rapidity of ulceration is the salient feature.

Tubercular ulcerating syphiloderm generally shows grouped ulcers, which are small, and often associated with

nodules. Here again the spread is more rapid. In both these types the effects of anti-syphilitic treatment can be tried, if doubt still remains.

(c) *Lupus vulgaris*.—Ordinary lupus often develops at a younger age than rodent ulcer, and epithelioma may follow prolonged X-ray treatment for this condition. In the surrounding parts of lupus vulgaris, there are usually "apple jelly" nodules, seen after compression with the diascope, which are so characteristic. Perhaps one of the most important features with us is the very rare occurrence of this disease in Australia.

(d) *Epithelioma*.—This is considered under epithelioma.

(e) *Milia*, such as occur in multiple rodent ulcer, have already been described, but they sometimes occur independent of this disease, and are for the most part grouped around the eyes, generally at the inner canthi. They show no tendency to break down and ulcerate.

(f) *Non-pigmented moles, fibromata, and warts* have to be distinguished from the early nodular stage of rodent ulcer. Here the history, onset, mode of development and appearance of the surface have to be considered, and, if necessary, a histological examination can be made.

(g) *Epithelioma adenoides cysticum* (Brooke).—This has been considered separately owing to the close resemblance of this condition to rodent ulcer, and to the divergence of opinion as to whether the lesions can break down in this condition.

PROGNOSIS

Is good, with the exception of the terebrant type of cases, and those involving the mucous membranes.

TREATMENT

Radium was at its inception announced by some to be a panacea for all ailments, an expectation hardly to be realised. However, it remains to-day one of our chief weapons in the destruction of various malignant diseases of the skin. Treatment by radium has in my hands met with marked success, and the cosmetic results are always good. It has an advantage over the Röntgen rays, in that it is more readily applied, as there is no cumbersome apparatus to adjust. Radium is a constant factor, whereas the vacuum of the X-ray tube is constantly changing. Its effects are more beneficial than the X-rays. When the new growth is located in the skin, radium is almost certain to destroy it, for its action is more destructive on the new epithelial growth than on the fibrous tissue. Thus it destroys the epithelial proliferation, and allows the underlying fibrous tissue to scar over. That fibrous tissue is very resistant to the action of the rays, and allows this to take place, is seen in a growth of this nature, viz. keloid, for the removal of which long unscreened applications are required. When subcutaneous fat and bone are reached treatment is not so successful. Recurrences after radium do sometimes occur, but are rare. Some of the most difficult early cases to cure are those affecting the conjunctival mucous membrane, for often in these one is unable to bring the radium into direct contact with the growth.

In the treatment of cutaneous lesions, radium is usually applied on a flat varnished applicator, which can be made in various sizes and shapes. The success attained with it depends largely on the experience of the physician. No hard-and-fast rule can be laid down, and the treatment of each case must be determined by its clinical

appearance. Apparatus appropriate for the removal of these growths are those containing pure, half-pure, and quarter-pure radium bromide, the first having a radio-activity of 2,000,000. Emanating from the radium are rays showing varying powers of penetration, of which the soft have but little penetrative action and are absorbed by the surface layers of the skin. The harder rays have a greater power of penetration and affect the deeper tissues. For practical purposes it is often necessary to eliminate the soft, or those rays which would have a burning action on the epidermis. With this object in view metal screens, composed of some metal such as nickel or lead, are used, which vary in thickness from one to one-tenth of a millimetre. To prevent any of the rays being occluded it is necessary to remove crusts before commencing treatment, and this can be accomplished with a starch poultice. When the growth is extensively elevated curetting may be an advantage, but this procedure is by no means essential. A week after treatment is commenced, there is usually some inflammatory reaction, but the destructive effect of the rays on the new-formed tissue continues for some weeks, when a crust forms on the lesion and leaves, after it falls, a fine, atrophic scar.

The Röntgen rays.—Röntgen rays can perhaps be used most advantageously in those extensive growths of the terebrant type, where the area to be treated is too large for the radium applicator.

Excision.—A successful result can be attained if the growth is freely excised, but at least half an inch beyond the margin of the ulcer, with a corresponding depth, must be removed. The cosmetic results following excision are inferior to those obtained with radium. Excision is often impossible when the ulcers are situated in the region of the

eye (a frequent site of rodent ulcer). The resulting scar following operation for rodent ulcers about the lower lid may lead to ectropion. Statistics show that excision is more often unsuccessful than not, and recurrence takes place. When this occurs rapid extension always follows, for the fibrous tissue barrier is broken down. I recall to mind the case of a patient, who had been operated upon three times unsuccessfully in one year, so that it is not surprising to find that many patients absolutely refuse operative treatment. I have seen recurrences several times in the orbit following enucleation; here the growth soon involves the fat, and then progress is rapid.

Carbon dioxide snow.—Freezing by this has met with success, and can always be applied to early or superficial types of rodent ulcer. Graham Little, after using this method somewhat extensively, has been impressed with his results.

Ointments.—The use of these is to be deprecated, as they have no influence on the progress of the growth.

PLATES TO ILLUSTRATE RODENT ULCER (FIGS. I TO 19)

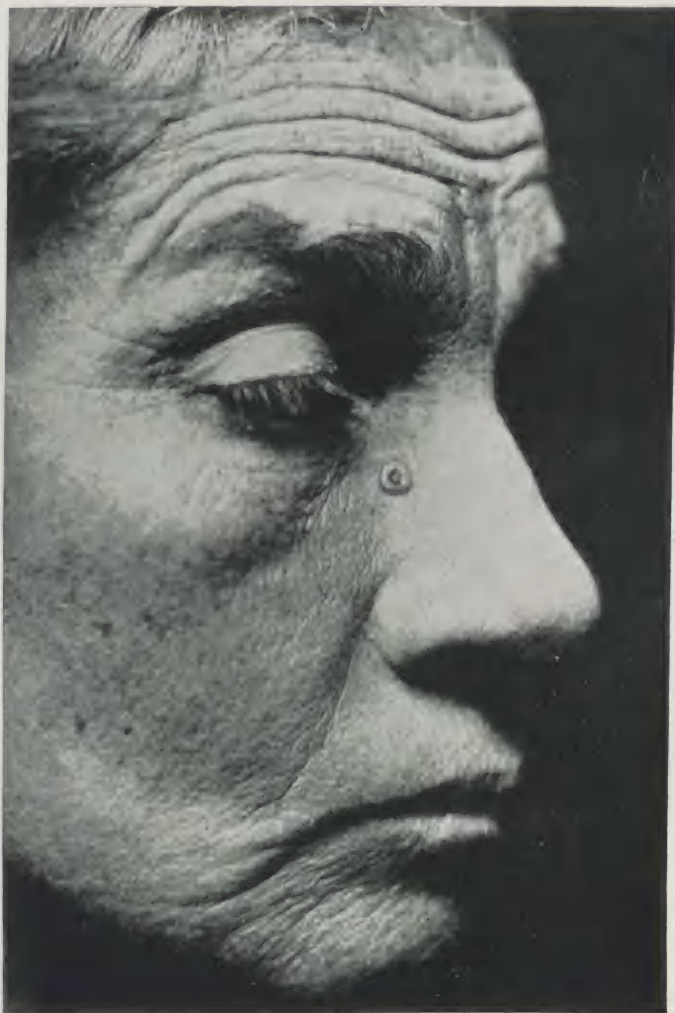


FIG. 1.—RODENT ULCER (Early)

Æt. 59. Early rodent ulcer with commencing ulceration.



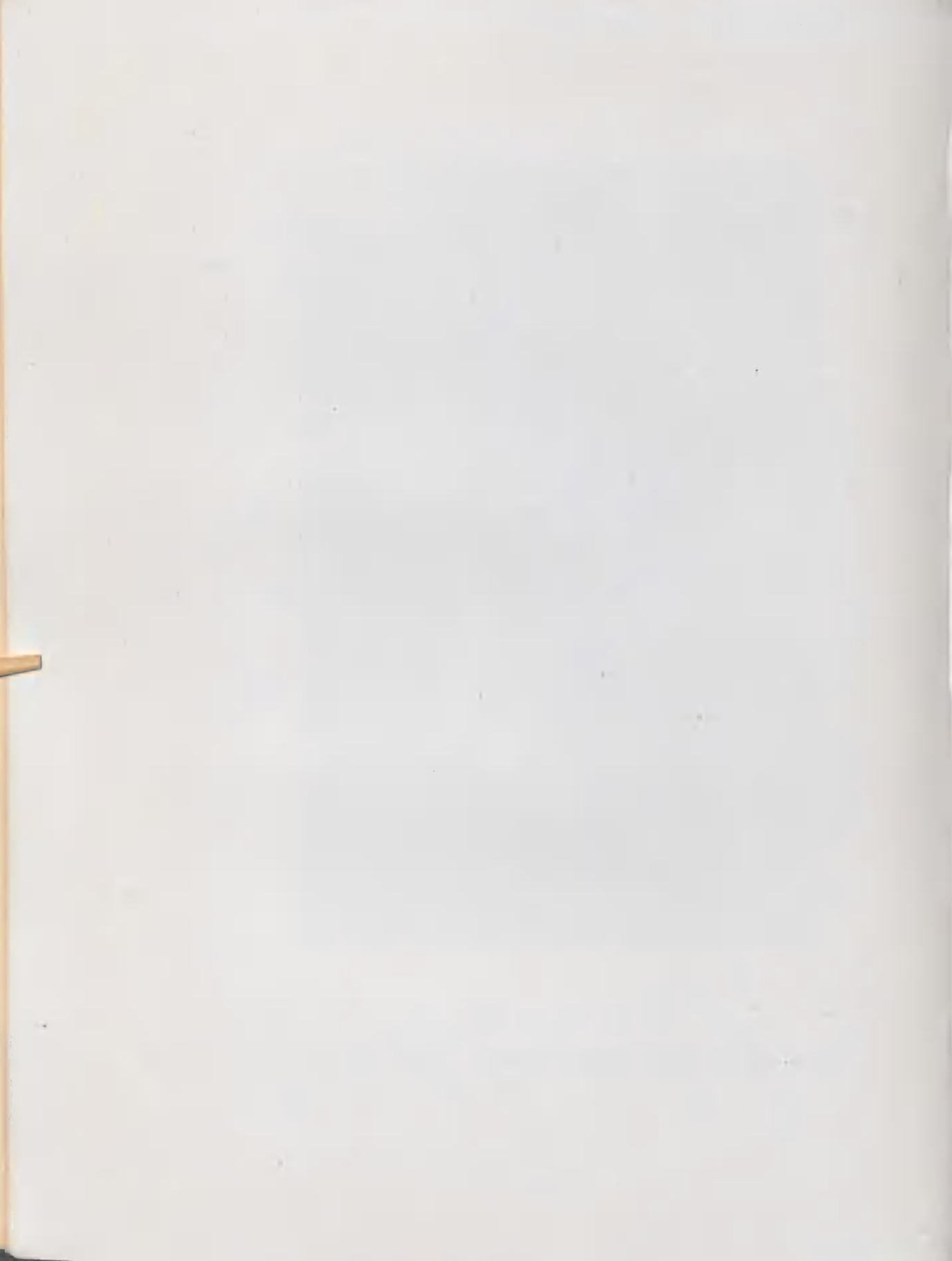
FIG. 2.—RODENT ULCER (Terebrant Type)

Æt. 48. History of starting as a pimple. The patient was later operated upon, when the growth spread rapidly.



FIG. 3.—RODENT ULCER

Æt. 51. Affecting the inner canthus of the right eye. Duration eighteen months.
Beneath the site a lesion appeared five years ago, but was cured by radium.



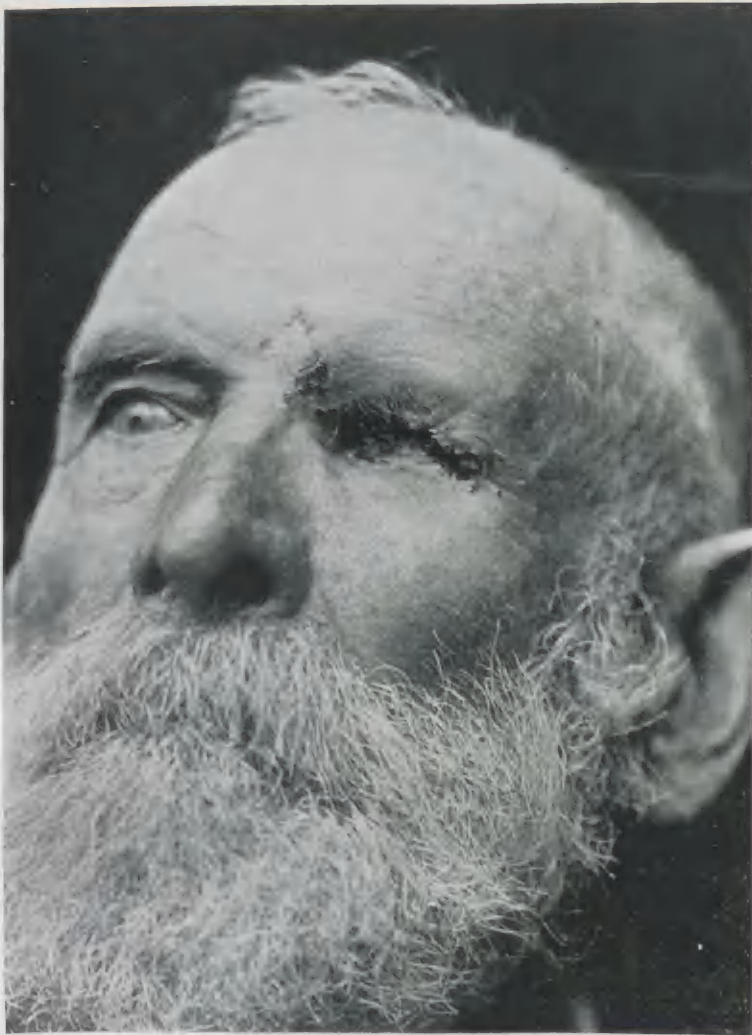


FIG. 4.—RODENT ULCER

Æt. 83. Duration twenty-one years. Treated for many years with lotions and ointments. Four years ago, when the eye was extensively affected, he commenced treatment with radium. The eye has never been enucleated, but the shrivelled remains are still present.

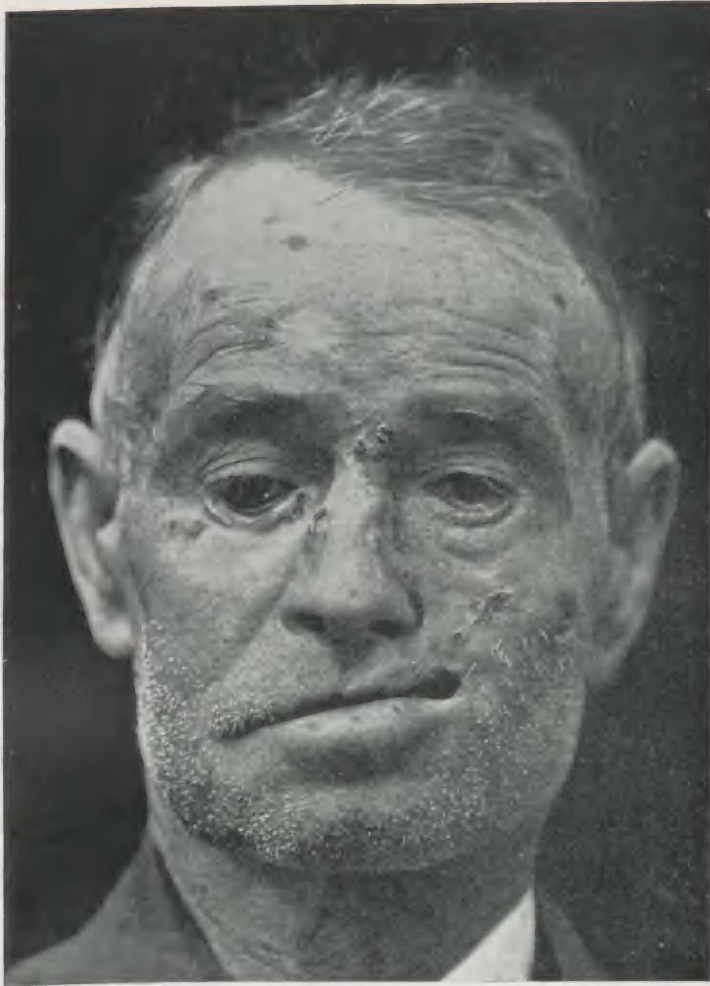


FIG. 5.—RODENT ULCER

Æt. 73. The lesion affecting the left angle of the mouth is of fifteen years' duration, and that affecting the left eye is of seven years' duration. The latter was a deep ulcer and covered an area now represented by the white scar. These two lesions were treated twelve months ago with radium, and the patient told to return in eight weeks for further treatment, which he neglected to do. He has again returned for radium treatment with several fresh rodent ulcers, and the incompletely cured old lesions.



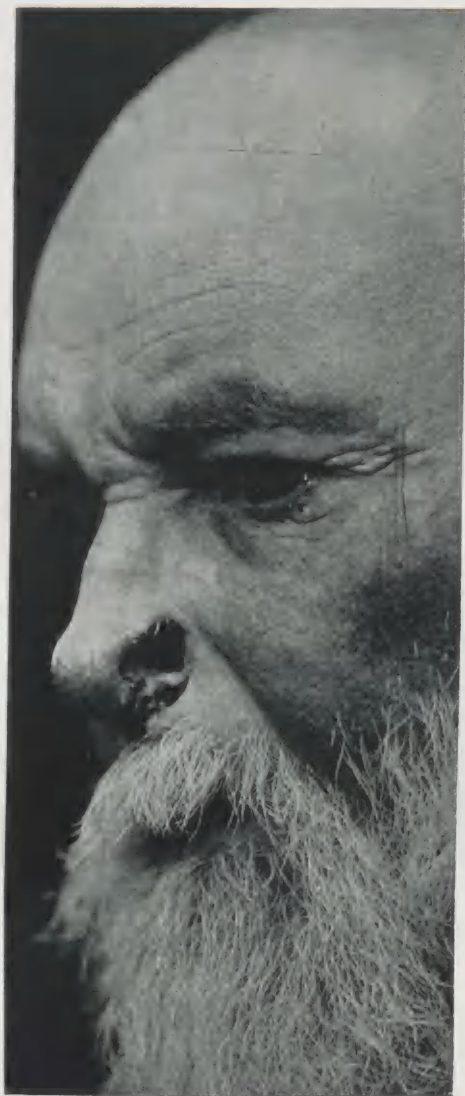


FIG. 6.—RODENT ULCER

Ætā 78½ Duration fifteen years.

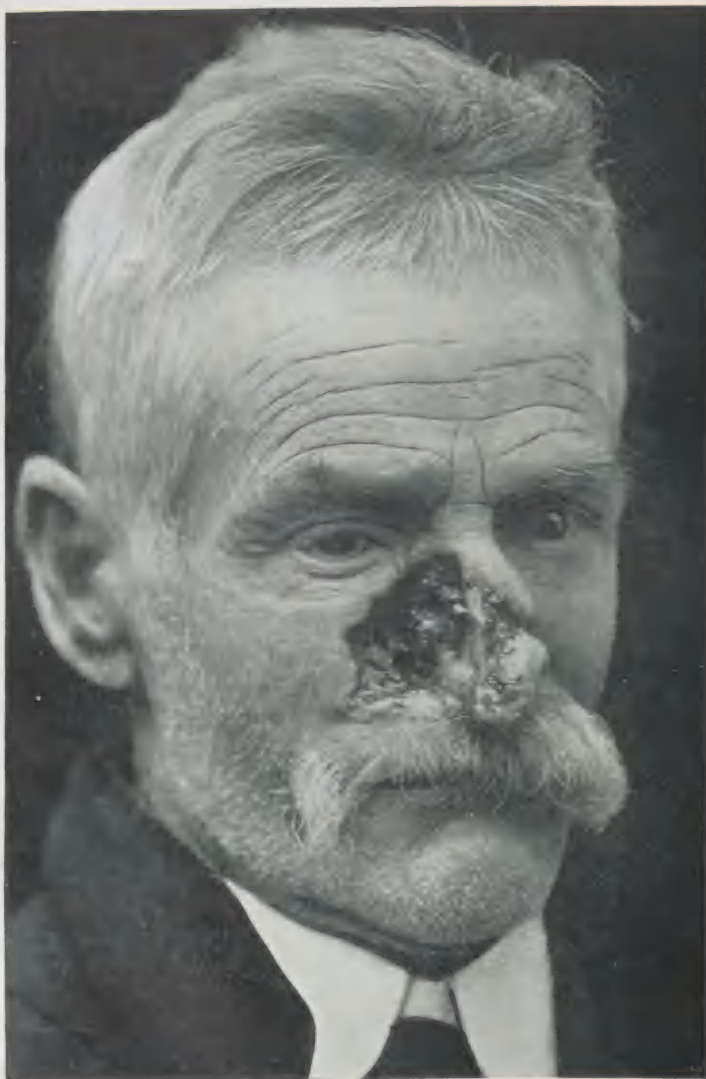


FIG. 7.—RODENT ULCER (Terebrant Type)

Duration seventeen years. Operated upon three times in the last five years. The patient was last operated upon twelve months previously. Since the operations the growth has spread rapidly.



FIG 8.—RODENT ULCER

Æt. 42. Duration two and a half years. History of starting as a mole, similar to those adjacent.

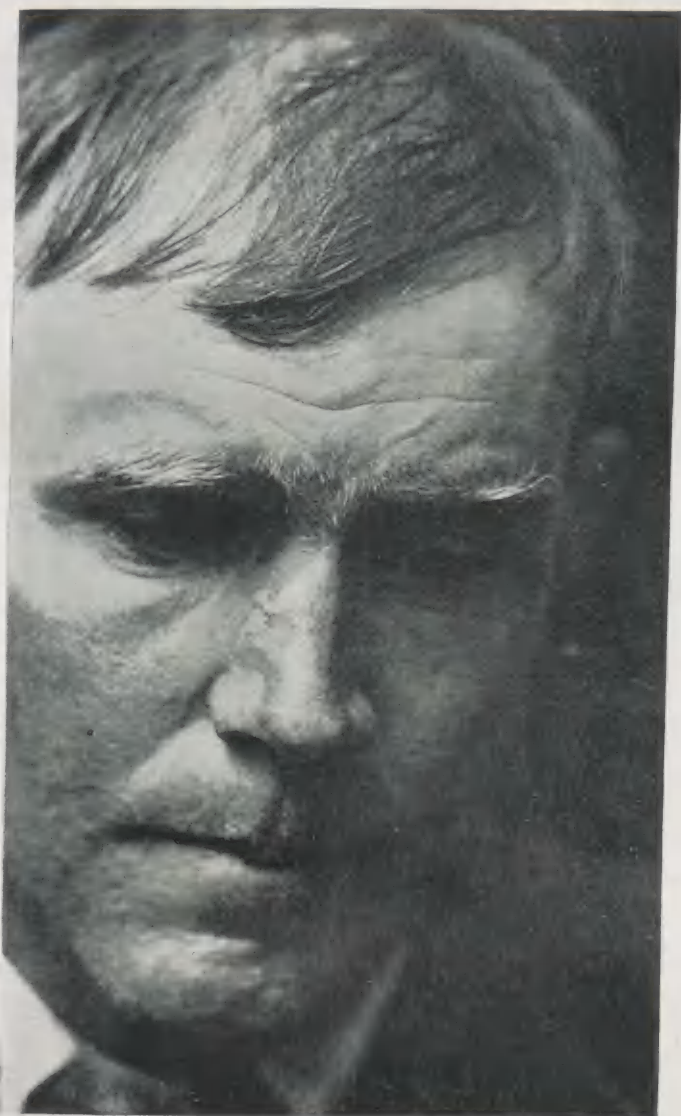


FIG. 9.—DIFFERENTIAL DIAGNOSIS FROM RODENT ULCER.
LUPUS ERYTHEMATOSUS

Of twelve months' duration. It shows an erythematous border, and
at one portion of the periphery sebaceous plugs.



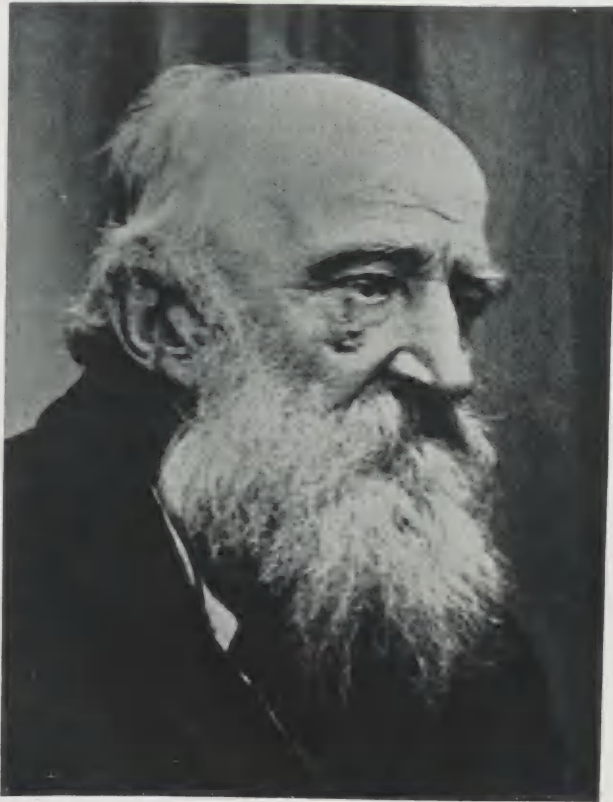


FIG. 10.—RODENT ULCER

Æt. 75. Duration three years. The lesion started after an injury from a hatpin.
Between the fingers it gave the impression of a coin being felt through a cloth.

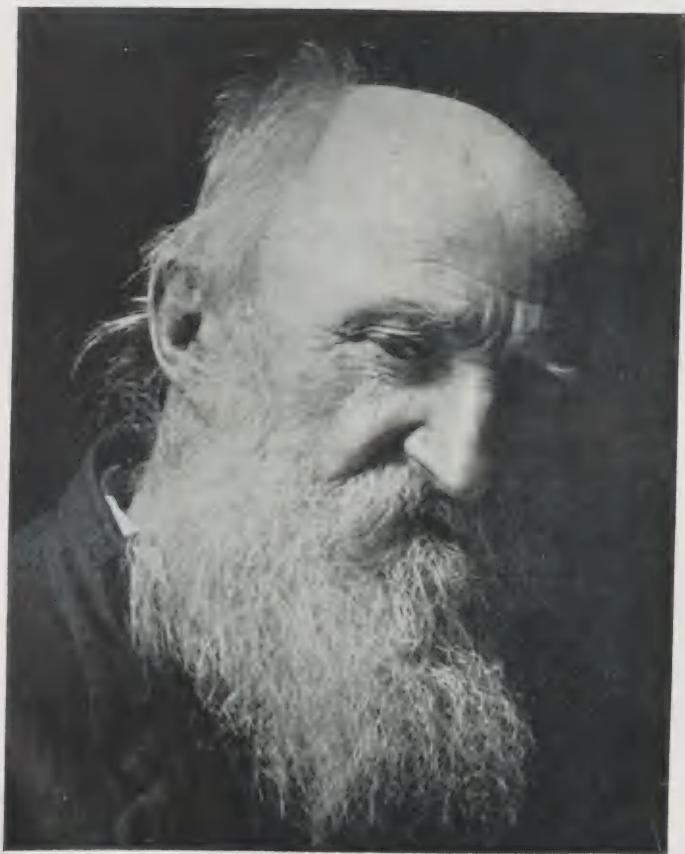


FIG. 11.—RODENT ULCER

After treatment with radium.

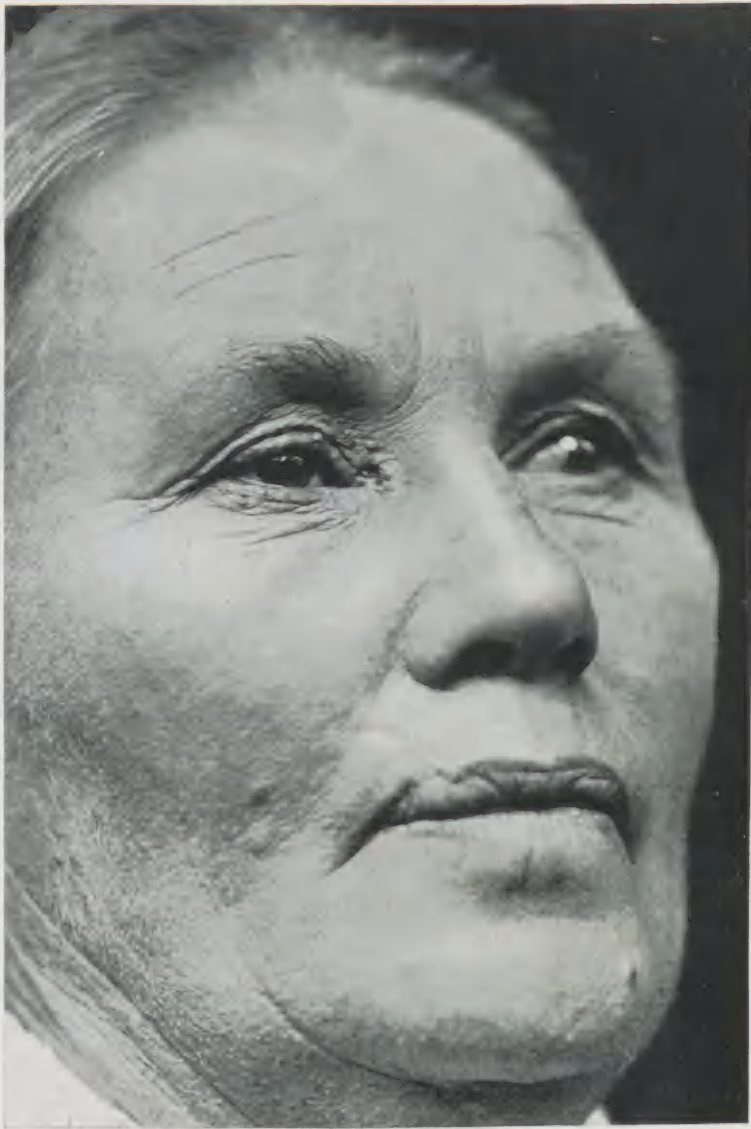


FIG. 12.—RODENT ULCER

Æt. 17. Affecting inner canthus of right eye. There is a well-marked beaded border.





FIG. 13.—RODENT ULCER

After treatment with radium.



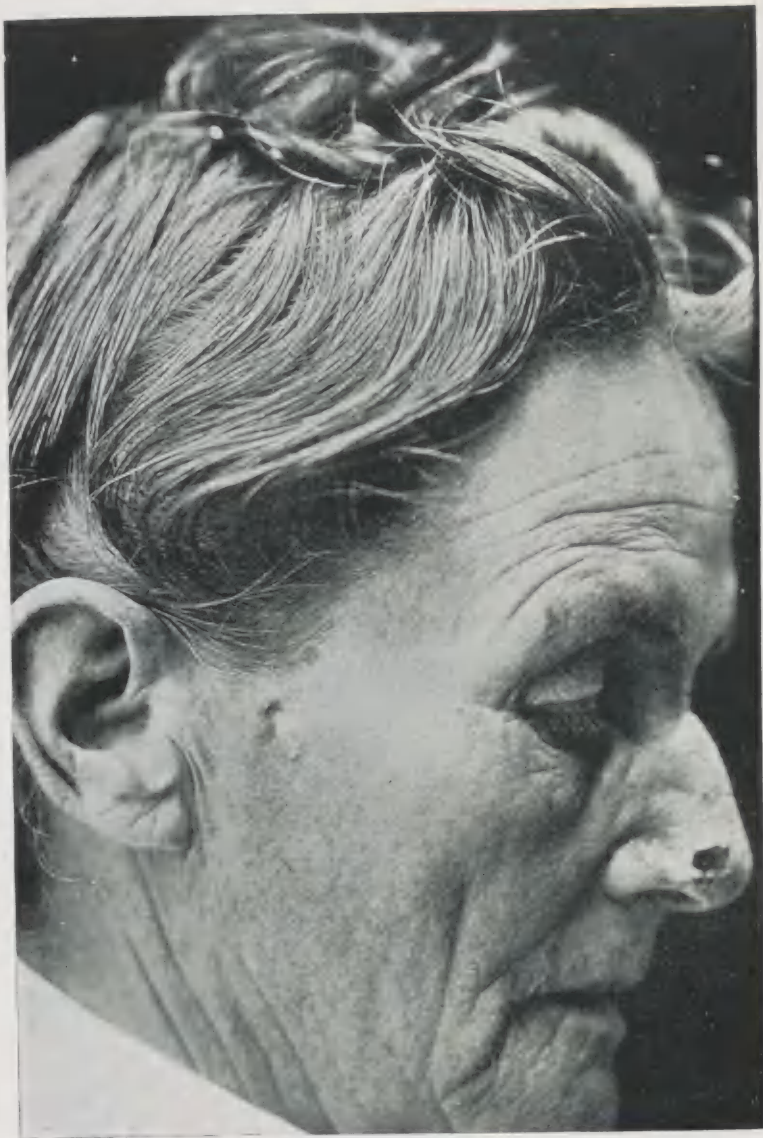


FIG. 14.—RODENT ULCER

Æt. 60. The lesion on the right side of the face has been present for many years and according to the history first manifested itself following upon a furuncle. The lesion on the nose has been present for eighteen months.

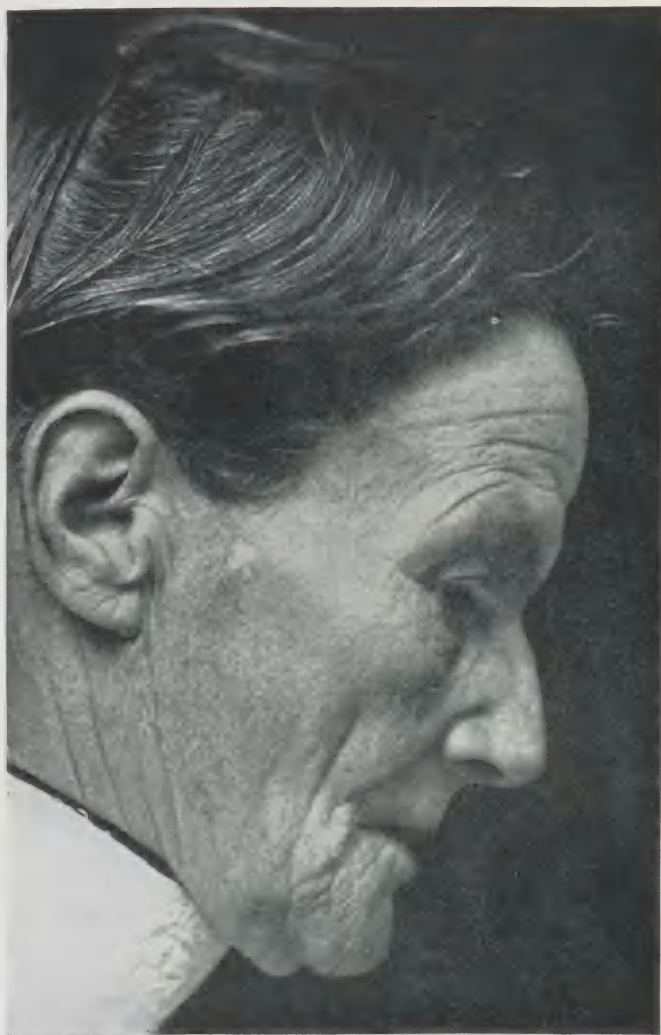


FIG. 15.—RODENT ULCER

After treatment with radium.



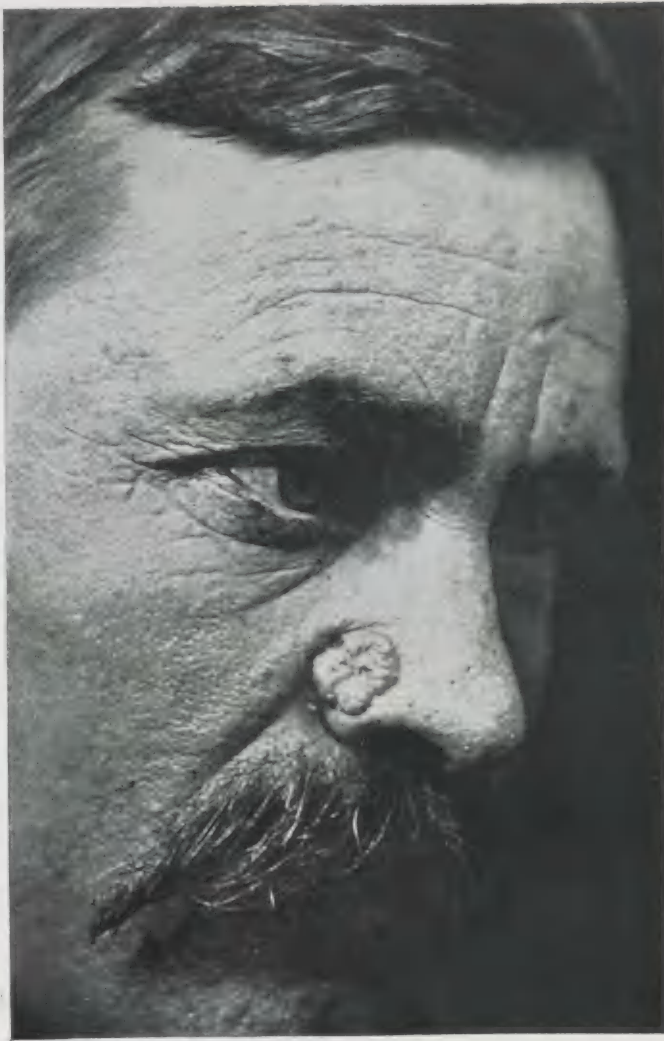


FIG. 16.—RODENT ULCER

Duration seven years. "Started as a flat warty growth." There is now a thick raised and rolled border, which is somewhat nodular, with dilated vessels coursing over it. Kerasinoses are absent.

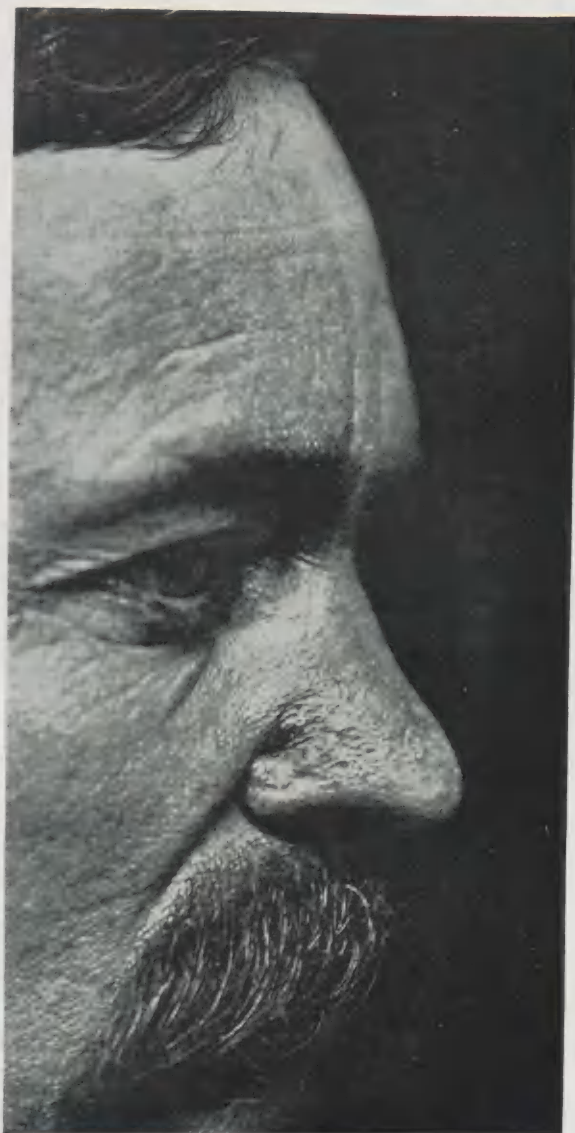


FIG. 17.—RODENT ULCER

After incomplete treatment with radium, the patient having
to leave for another State.

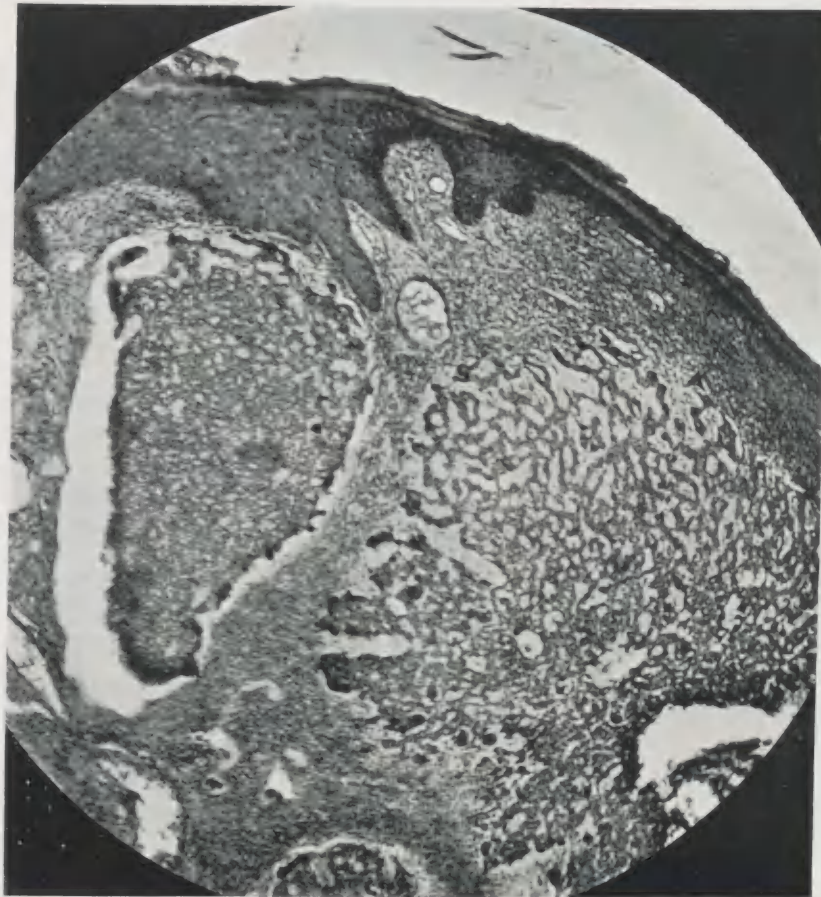


FIG. 18.—RODENT ULCER

Micro-photograph of section of rodent ulcer, Fig. 16.

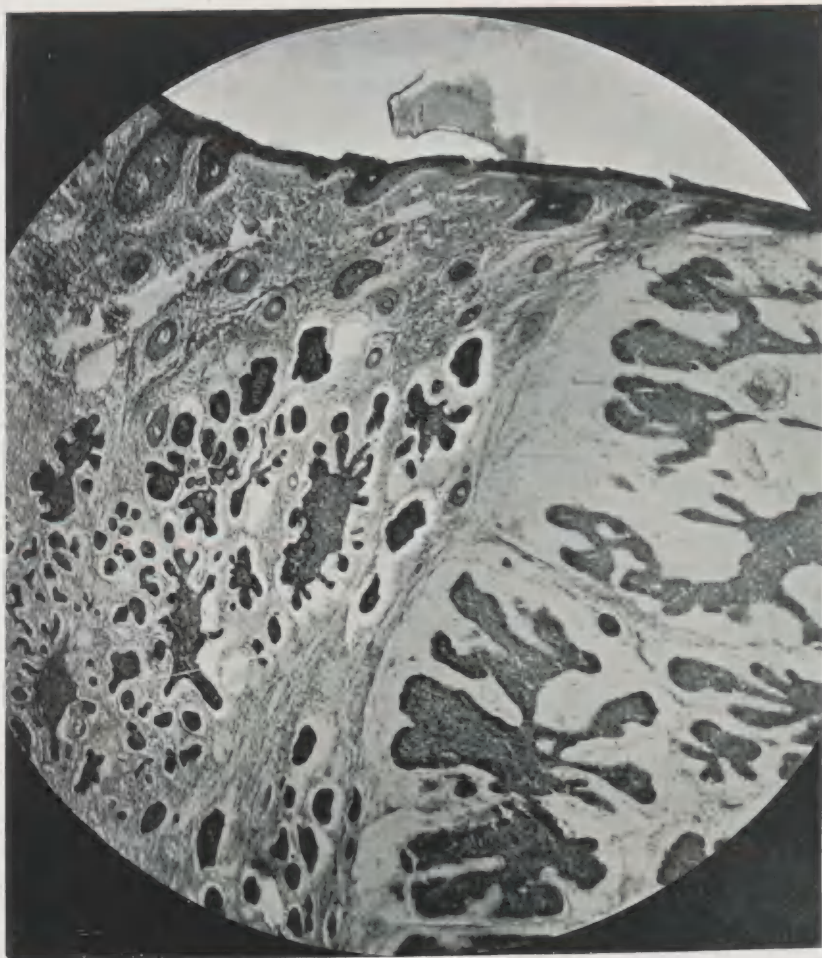


FIG. 19.—RODENT ULCER

Micro-photograph of a section of a nodule from the face. There is no evident connection of the growth with the surface epithelium, which appears to arise from the hair follicles. The right half of the growth shows degeneration of the connective tissue.



DERMATITIS SOLARIS CHRONICA

DEFINITION

The term *dermatitis solaris chronica* will be used to designate a degenerated condition of the skin, appearing only upon exposed parts, and particularly associated with advancing years. This degeneration, however, is proportionate not to years, but rather to the exposure to sunlight. It is characterised by erythroderma, freckle-like spots or areas of brownish pigmentation, telangiectases, white sclerotic spots, and keratoses, which may ultimately develop into epitheliomata. All these manifestations constitute the varied attempts of the skin to destroy the injurious influence of light.

ETIOLOGY

Strong and prolonged sunlight, acting upon a skin which has undergone senile changes, is the most potent factor in its production. The condition is one of great frequency in Australia.

SYMPTOMS

The disease is localised to exposed parts, such as the face, neck, dorsum of the hands, and the forearms. In a well-developed case all the lesions may be present at the same time. These include (1) erythroderma. (2) Scattered pigmented spots or areas, of various sizes, and of a pink or brownish colour. These pigmented patches are amongst

the first indications that the normal balance of wear and tear has been lost, and form an attempt to protect the underlying tissues from the actinic rays of light, which the brown colour of the pigment is able under normal circumstances to absorb. (3) If these patches of pigmentation fail to maintain this balance of tissue economy, then the epidermis proliferates as a further means of protection to the subjacent tissues. This proliferation may appear as a slight scaly roughness, or be more pronounced and appear as raised, rough, circumscribed, somewhat warty-looking lesions, the size of a split pea, or smaller, and either dry or greasy in appearance. It is upon the patches of pigmentation that these epidermal proliferations or keratoses most frequently, though not invariably, arise. Although these hyperkeratotic lesions are already described under various names, viz. senile or seborrhœic keratoses, it would in the writer's opinion be more appropriate to designate them keratoses solares, for their existence only on exposed parts demonstrates that they are not due merely to the senility of the skin. Under this nomenclature, seborrhœic warts (*verruca plana senilis*), which are also present upon covered parts, especially the back, etc., should not be included, but should be looked upon as a separate and independent condition. They are considered by Unna to be of nævoid origin (*nævus seborrhœicus*). Some of these keratotic lesions are dry and of a greyish colour, whilst others are greasy, and of a greyish to greyish black colour. The latter type occurs on the face, where the sebaceous glands show increased activity early, and here sebaceous material may enter largely into the composition of these keratoses at their inception. The sebaceous material, like the pigment, appears to be thrown out for protective purposes, and may even tend to form diffuse patches. In this stage they

can readily be removed, and frequently show on their under surface spine-like processes, which have arisen from the pilo-sebaceous follicles. Beneath these, degenerative changes not infrequently take place, with the production of ulceration of the surface layers of the epidermis, whilst the deep layers show proliferative changes. In these malignant ulcers progress is rapid, especially if pus be allowed to collect beneath them. These keratoses are often described as being the starting-point of rodent ulcer, but this is unusual, for they are generally the precursors of epitheliomata, rarely of cutaneous horns. (4) Sometimes these epidermal thickenings fall off, and leave white atrophic scars. (5) Telangiectases are also to be noticed distributed over the affected surface.

PATHOLOGY

With marked *dermatitis solaris chronica*, the skin in general presents those changes which we are accustomed to see in the senile skin. These are represented by a degeneration of the elastic and collagenous fibres of the corium, with some scattered cell collections, and dilatation of the capillaries. There is some shortening of the hair follicles, together with dilatation of the sebaceous glands, and the fat may be absent, or decreased in quantity. The epidermis is in general thinned, and the brown patches are represented by accumulations of pigment mainly in the deeper layers of the epidermis, both intra- and extra-cellular, as well as in the upper portion of the corium. The telangiectases are due to the compensatory dilatation of some of the vessels that results from the disappearance of others.

Keratoses.—An outstanding feature in sections is the thickening of the horny layer of the epidermis, some of

the cells undergoing imperfect cornification, and retaining their nuclei (*parakeratosis*). The mouths of the pilosebaceous follicles are patulous, and filled with horny plugs. The prickle-cell layer exhibits marked variation. Whilst the early lesions show but slight increase, all gradations of hypertrophy of this layer (*acanthosis*) up to actual infiltration of the corium are to be seen. The papillæ in the early stages are largely obliterated. The corium shows cellular infiltration, which becomes marked when prolongation downwards of the interpapillary processes takes place. R. L. Sutton states¹: "The growths could be separated into three distinct groups, first, and most frequent, a keratoid variety, characterized by great corneous hypertrophy, with some parakeratosis, a moderate degree of acanthosis, slight proliferative changes in the germinal layer, and more or less flattening of the papillary bodies; second, a nevoid type, which was practically identical with Unna's 'nævus seborrhœicus'; and third, an acanthoid or verrucose form, distinguished by considerable hyperkeratosis, very pronounced acanthosis, signs of exceedingly active proliferative changes in not only the stratum germinativum, but also at numerous other points in the rete, and enormous papillary hypertrophy. In these wart-like specimens, the cutis showed inflammatory changes of a subacute nature, with capillary dilatation, extensive perivascular infiltration, small collections of leukocytes, and round and plasma cells scattered through the upper derma. . . . The sebaceous glands were practically normal in all three classes, despite the fact that all contained considerable quantities of free fat. The nevoid tumours were richest in this material, the acanthoid less so, and the keratoid least of all." That sebaceous material enters

¹ *Journ. Amer. Med. Assoc.* 1915, lxiv. pp. 405, 406.

into the composition of some of the earlier lesions can be demonstrated by staining with osmic acid, the mass immediately becoming black.

DIAGNOSIS

The various lesions appearing together on exposed parts in those usually past middle life renders the diagnosis easy. The keratoses are readily recognised from ordinary warts by their rough scaly appearance.

PROGNOSIS

The keratoses may fall off, and leave indefinite atrophic scars. Often they remain stationary for years, but not infrequently they develop into epitheliomata, and on this account lesions of this nature should receive treatment.

TREATMENT

Ointments with keratolytic action may remove the hyperkeratotic lesions in the early stages, but the lesions generally recur. Their destruction can be attained by the application of radium or the X-rays. They can also be excised, or carbon dioxide snow may be applied. Large hats for men and brown veils for women would afford a large measure of protection to the face.

PLATES TO ILLUSTRATE DERMATITIS SOLARIS CHRONICA
(FIGS. 20 TO 25)

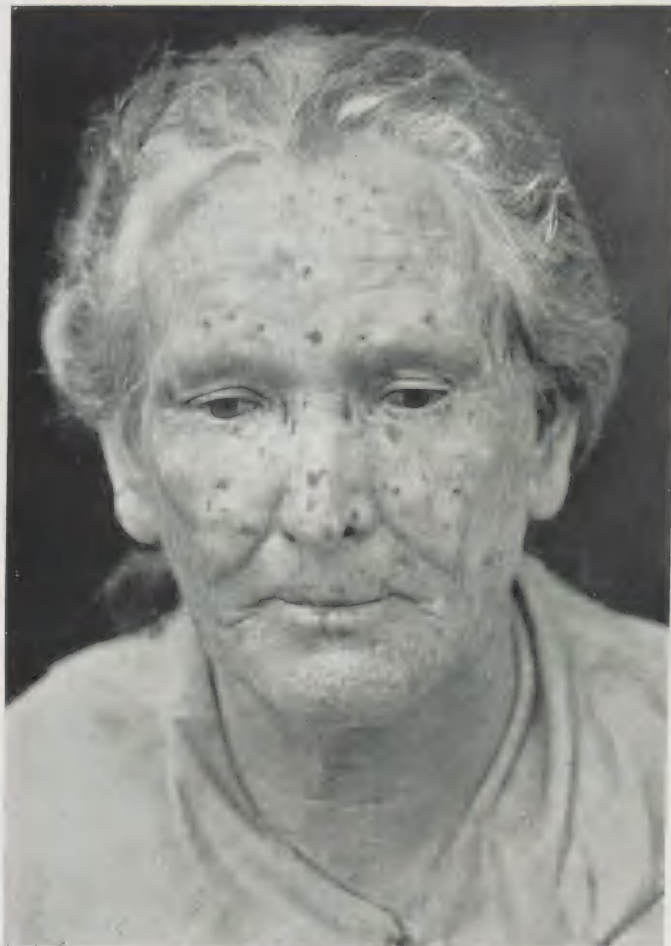
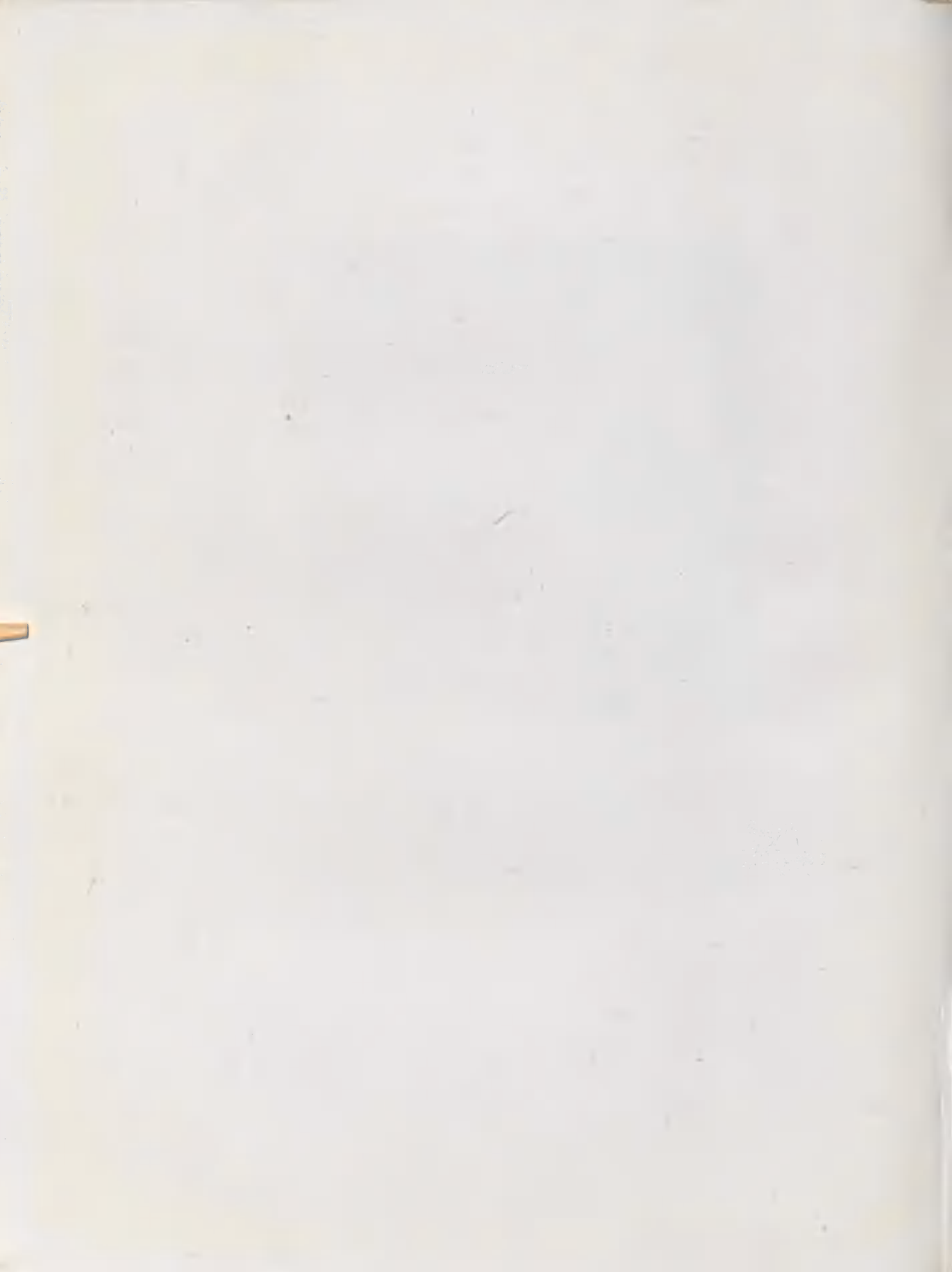


FIG. 20.—DERMATITIS SOLARIS CHRONICA, SHOWING KERATOSES

Æt. 61. Duration thirty years.



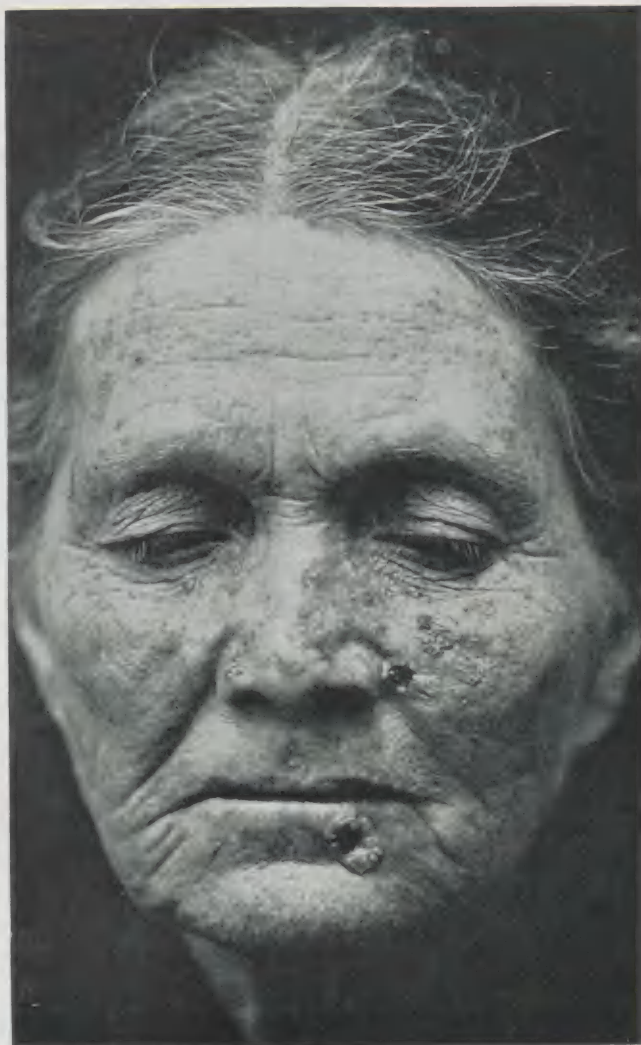


FIG. 21.—DERMATITIS SOLARIS CHRONICA, SHOWING MULTIPLE
KERATOSES AND EPITHELIOMATA

Æt. 66. The history given is that the ulcers have only been present three weeks. Probably they have arisen beneath greasy keratoses, ulceration being well advanced before the crusts have dropped off.



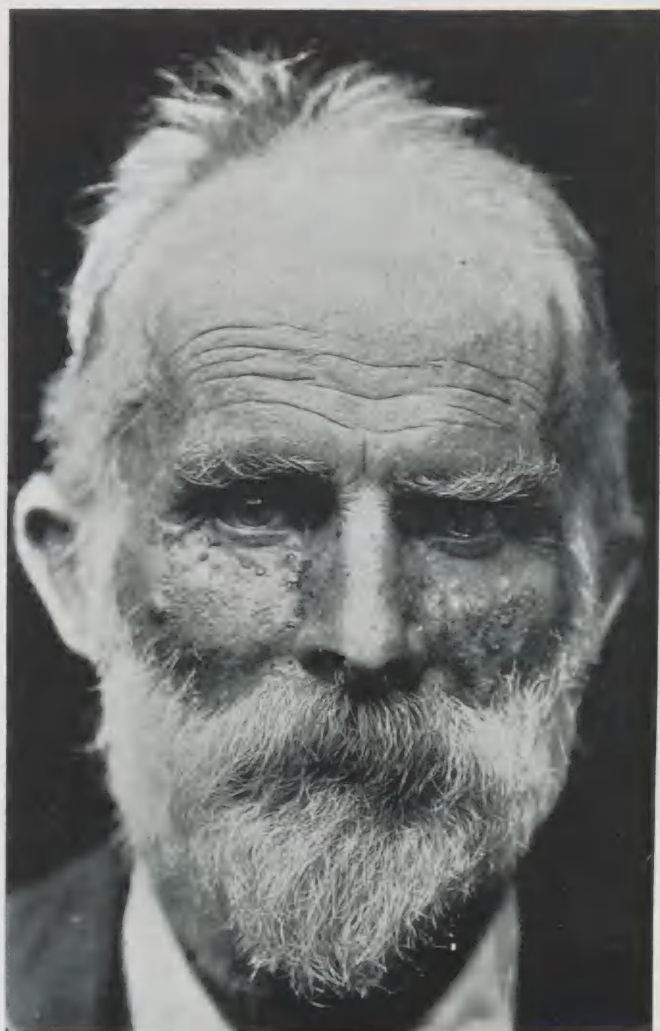


FIG. 22.—DERMATITIS SOLARIS CHRONICA, SHOWING MULTIPLE KERATOSSES ON FACE, AND EPITHELIOMA OF NECK

Lesions such as these on the face can mostly be removed with keratolytic ointments, but they tend to recur. Same patient as shown in Fig. 23.



FIG. 23.—EPITHELIOMA OF NECK

This is the same patient as Fig. 22, showing the face with multiple keratoses.

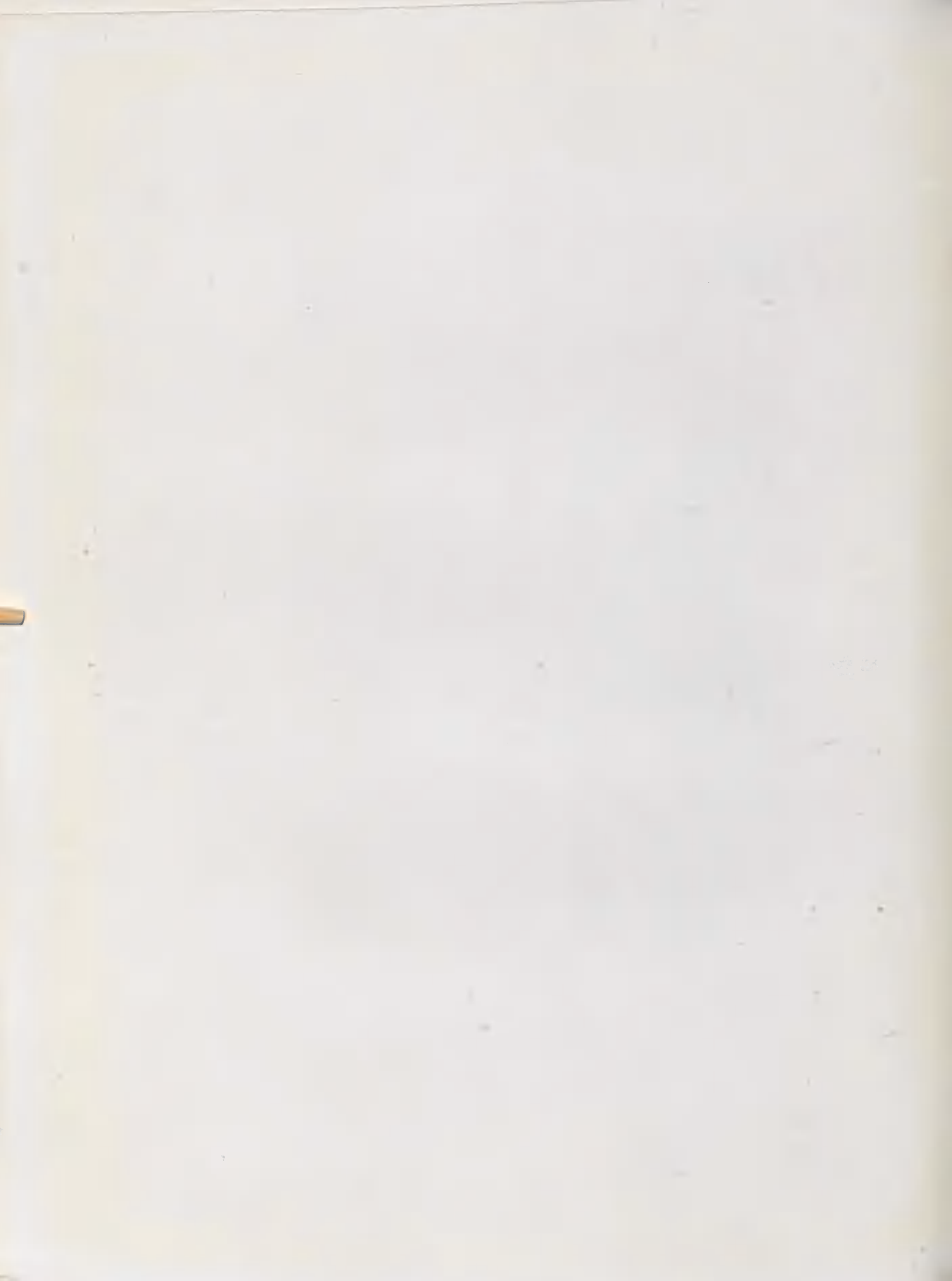




FIG. 24. DERMATITIS SOLARIS CHRONICA, SHOWING KERATOSES AND
EARLY EPITHELIOMATA

Æt. 77. The epithelioma on the lower part of the face was covered with a large crust. There was an extensive thin crust behind the ear, beneath which was also a small epithelioma. The small raised circumscribed growth just exterior to the eye was removed for pathological examination. See Fig. 25.



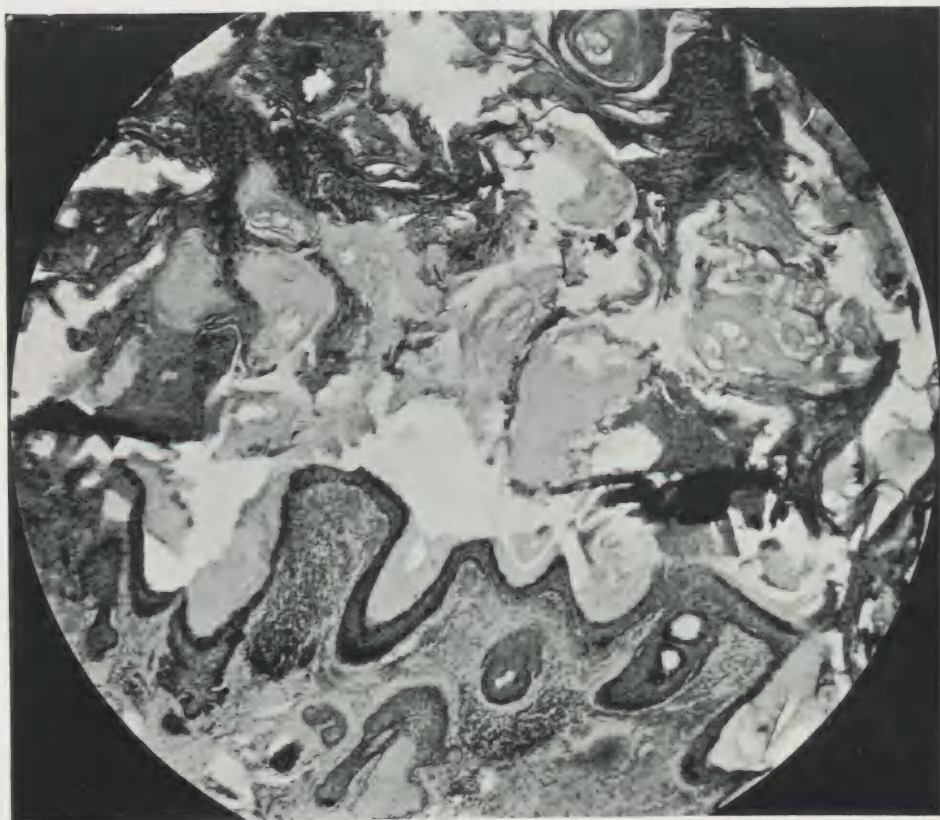


FIG. 25.—KERATOSIS SOLARIS. Micro-photograph of section from Fig. 24.

The stratum corneum is greatly thickened, many of the cells having retained their nuclei (parakeratosis). Sebaceous materials also aid in this thickening, whilst the pilosebaceous openings are patulous. The portion of the corium beneath the keratosis exhibits a small cell infiltration which is not marked, but is an early evidence of malignancy.



CUTANEOUS HORN

DEFINITION

A true horny outgrowth from the epidermis.

ETIOLOGY

Cutaneous horns are stated to arise mostly from sebaceous cysts, warts, and scars. Unna asserts that many clearly develop from dermoids and atheromata, and rejects the view that they arise from sweat glands. The history usually given by patients is that they start as warty growths, doubtless from keratoses. This would account for their presence most frequently in those past middle life. Lebert, in a review of 109 cases, found authentic evidence of their origin from a warty spot in twenty-seven cases. Females appear to be more frequently affected than males.

SYMPTOMS

Cutaneous horns, which are generally single, but may be multiple, are softer in consistence than those of animals. They appear as solid, dry, somewhat conical outgrowths, variable in size and shape. They may be straight, or curved, round, oval, or angular on section, and of a greyish, greyish brown, or greyish black colour. Longitudinal furrows are seen on the surface. The sites of predilection, excluding the genital regions, are those exposed to weather, viz. the face, dorsum of the hands, and the forearms.

Occasionally, there is to be observed on the nose a thin filiform type of cutaneous horn, originating almost invariably from a keratosis, but the larger sized horns mostly appear on the dorsum of the hands and the side of the face, and are commonly associated with *dermatitis solaris chronica*. The latter type show an elevation and broadening of the soft tissues at their proximal end. The freedom of movement of the horn causes constant irritation to its base, and this, if prolonged, is liable to produce epitheliomatous changes. Lebert's investigations show that 12 per cent. develop into epitheliomata. This change occurred in a case recently seen by me, where the lesion was situated on the dorsum of the hand, and was loosely attached in the centre of a fungating epithelioma. This is not an improbable result when they develop, as they so frequently do, from keratoses, which are themselves to be considered a degenerated or precancerous condition of the skin. Cutaneous horns, if cast off, usually reappear. Their progress is slow and unaccompanied by symptoms.

PATHOLOGY

The early histological structure shows a proliferation of the prickle-cell layer (*acanthosis*), as well as an increase in the horny layer (*hyperkeratosis*), thus resembling an ordinary wart. Taps of epithelium, constituting the interpapillary processes, grow down into the corium, and the papillæ become long and narrow. It is upon these elongated papillæ that the growth is built up. The granular layer is hypertrophied, whilst the small vessels of the papillæ are dilated. Later, horn formation takes place rapidly, many of the cells retain their nuclei (*para-keratosis*), thus accounting for their abnormal cohesion.

PROGNOSIS

The condition is one that must receive immediate attention, owing to the probability of malignant changes.

TREATMENT

Under no circumstances should acids or alkalies be applied, or similar methods which are liable to irritate, without causing complete destruction of the growth. The malignant changes in the case referred to above could be attributed to the application of acids. The small filiform horns can be snipped through at their base with scissors, and then radium applied, or they may be excised. Better cosmetic results are obtained with the former method. The large cutaneous horns should be excised, and radium applied as a precautionary method against recurrence.

PLATES TO ILLUSTRATE CUTANEOUS HORN
(FIGS. 26 AND 27)



FIG. 26.—CUTANEOUS HORN

The face of the patient exhibits various sized pigmented patches, some of which are the size of a threepenny piece. In colour they are yellow, brown, brownish black, or even a pinkish colour. The smaller pigmented spots for the most part show no thickening, but there is a scaly thickening in some of the larger red and brownish pigmented patches. Keratosis have been present on the nose for the past five years, but occasionally some fall off. On the nose the pilo-sebaceous follicles are patulous. The cutaneous horn is three-quarters of an inch in length, with four surfaces, the superior and inferior of which are slightly broader than the lateral surfaces. The horn was situated on a fleshy base, and was freely moveable. It was first noticed six months ago.



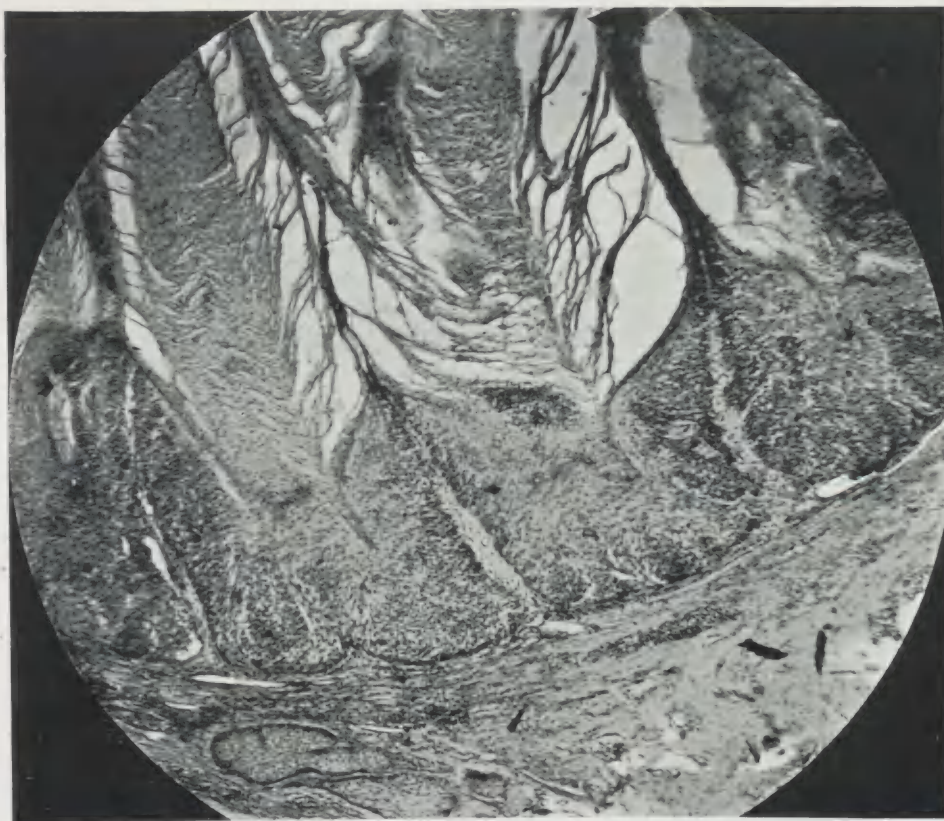


FIG. 27.—CUTANEOUS HORN

Micro-photograph of a section from the base of the cutaneous horn seen on the face in Fig. 26. The upper portion of the photograph shows the horny substance of the growth, composed of horny cells many of which have undergone imperfect cornification, and retain their nuclei (parakeratosis). Beneath this is to be seen the greatly hypertrophied prickle cell layer, in which the papillae are elongated and narrowed. Many of the cells forming the lower strata of this layer are small and closely packed together, doubtless representing the actively proliferating cells, from which the prickle cells are destined to arise. Beneath this is a dense layer of fibrous connective tissue, encapsulating and encircling the growth.



MULTIPLE RODENT ULCER

DEFINITION

Under this title will be included those cases which are widely distributed over the face, and exhibit multiple and numerous lesions, simulating those of ordinary rodent ulcer.

ETIOLOGY

The condition is a rare one, only two cases of this nature having come under my observation in the past three years in the Radium Department of the Sydney Hospital. It appears to be of nævoid origin; possibly strong sunlight may be an exciting factor in its production. The treatment of a primary growth by Röntgen rays has been declared by some to stimulate fresh neoplasms to activity. In one of the above cases the Röntgen rays had not been applied until numerous growths had been discovered. Both patients were males, in whom the disease first manifested itself between the twentieth and thirtieth year of life, and they were the only members of the family to be affected. Some mental dullness or deficiency may be noticeable in these patients.

SYMPTOMS

The disease is localised to the face, to that portion of the neck adjacent to the posterior sulcus of the ears, and the meatus of the ear may show involvement. As the region of the eye is a frequent site for their occurrence, it

is only a matter of time before the eye itself becomes implicated. The individual lesions are for the most part discrete, and scattered freely over the face, without any marked tendency to grouping. When the condition has been present for some years, lesions in all phases are to be seen, from small nodules, through their various transitional stages, up to actual ulceration. The nodules vary from the size of pinheads to 5 or 6 millimetres, and after attaining a variable size they become umbilicated, and break down in the centre. There are usually fifty or more of these nodules present, which often present a pearly appearance; some, however, resemble the normal skin in colour. Ulceration may be either superficial or deep, as in ordinary rodent ulcer. A feature of some ulcers is that the raised border is absent, whilst others present this beaded border, with telangiectases or fine vessels coursing over it. This sign is also noticeable in the early nodules. Milia are not infrequently present, and evolve as smooth, round, pearly white, pinhead-sized lesions, which are non-inflammatory. They are for the most part distributed on the upper portion of the face in variable numbers, and are well portrayed on the forehead in Fig. 30. They appear insidiously, presenting no subjective symptoms, and often pass unnoticed by the patient. No opening can be seen on the surface, and after reaching millet-seed size, they tend to remain stationary. Milia are cyst-like structures, composed of several layers of epithelial cells. There is a diversity of opinion as to their site of origin, and various authorities have described them as arising from,

1. *Sebaceous glands*.—The milia of infants and those on the genitalia are regarded as retention cysts of these glands.

2. *Sweat ducts*.—Milia may be due to their dilatation,

and occur in such conditions as *epidermolysis bullosa*, and may be associated with scars (Darier).

3. *Hair follicles*.—These also are due to dilatation (Unna, Virchow).

4. *Embryonic germs of epithelium*.—Robinson of New York classifies milia as being of two kinds, viz. (a) milia without any exit or fat contents; they are considered as being derived from misplaced embryonic epithelium, from either the prickle-cell layer, or from a hair follicle. These he regards as true milia. (b) Milia with an exit, and with sebum and cholesterin, in short, a deep-seated comedo.

PATHOLOGY

Examination of sections from these growths reveals a microscopical picture, which is constant in appearance. In the corium there are masses of cells, many of which are lobulated, and show but little evident connection with the basal-cell layer. These masses are composed of closely packed epithelial cells, which are somewhat oval in shape, and have a well-defined nucleus, but little protoplasm. They are sharply circumscribed by a marginal palisade layer of cells. With ulceration of the surface of the nodule there may be observed in some of the more superficial of these masses signs of necrosis, as evidenced by the cells being but faintly stained. Between the new growth is a somewhat cellular and vascular connective tissue, whilst limiting the growth inferiorly is a more highly organised fibrous tissue. The hair follicles and the glandular elements of the skin are absent in that portion of the corium invaded by the new growth. There may also be present at the lateral borders of the growth some collections of small round cells. The papillæ are mostly obliterated,

and the epidermis may even appear to be thinned by pressure from beneath.

DIAGNOSIS

Epithelioma adenoides cysticum (Brooke) is a condition which bears a very close clinical and histological relationship to multiple rodent ulcer. It is a rare and benign nævoid condition, which often occurs in several generations. The disease as a rule commences about puberty, and there is often an hereditary tendency. Females are more prone to this affection than males. The eruption, which is mainly localised to the face, is symmetrical, and is especially grouped about the central perpendicular third, that is, around the nasofacial folds, the upper lip, chin, inner canthi, and the adjacent portion of the forehead. The region in front of the ears is commonly affected as well as the outer canthi. The lesions are for the most part discrete, firm, translucent, and vary from millet-seed size to lesions slightly smaller than hemp seed. A few may show telangiectases. There is no inclination to break down, or to spontaneous disappearance of the nodules. In a case that I saw lately, the father, grandfather, as well as two daughters and two sons, were affected. The grounds on which these closely allied conditions are to be differentiated are the following. Multiple rodent ulcer occurs at a later date, and has a tendency to break down. In *epithelioma adenoides cysticum* the distribution is more symmetrical, and localised to certain well-defined areas, whilst in multiple rodent ulcer the distribution is general. In the latter disease, its occurrence is more frequent amongst the male sex, as opposed to the disease being more frequent, but not entirely limited to the female sex in *epithelioma*

adenoides cysticum. The following differential diagnosis of cases has been made by Dr. Adamson :

CLINICAL DIFFERENTIATION

"In those styled Multiple Rodent Ulcer or of Multiple Rodent Ulcer Type.

1. All cases (with one exception, namely, White's case) occurred in adult males.

2. Not hereditary.

3. Distribution of lesions irregular.

4. Lesions of widely different sizes.

5. Tendency of lesions to increase in size, and to break down, *i.e.* to become locally malignant.

In Epithelioma Adenoides Cysticum.

1. All cases occurred in women, and the lesions appeared in childhood.

2. Generally in mother and daughter.

3. Distribution of lesions markedly symmetrical.

4. Fairly uniform size of lesions.

5. No tendency of the lesions to enlarge beyond the size of a split pea, nor to break down, *i.e.* to become locally malignant."

Histological Differentiation.—In both diseases "the lesion is made up of an epithelial growth, derived from the basallayer of the epidermis, or from that of the hair follicles. The growth is in the form of cell masses, with a marginal palisade layer, and central oval cells. There is a tendency to cyst formation in both, *i.e.* colloid cysts, and epidermal cysts (or *milia*). In both, the epithelial masses have new-formed encapsulating fibrous tissue. Here, however, the resemblance ceases, and we find that while the lesion of Brooke's disease is sharply circumscribed by a highly organised fibrous tissue, there is in the rodent ulcer a more highly cellular, and therefore more actively growing, fibrous tissue element, a plasma-cell exudation at the advancing margin, and outlying groups of epithelial cells invading the tissues beyond the main growth. These differences in the histological features correspond with, and explain to a

certain extent, the differences in the clinical behaviour of the lesions, namely, that one is restricted or benign in its growth, while the other tends to enlarge, and to involve and destroy neighbouring structures, or, in other words, is locally malignant. But of the essential cause of this difference in behaviour we are ignorant" (*Lancet*, 1908, ii. p. 1137).

PROGNOSIS

The outlook is not encouraging. Unless efficient treatment is persevered with, the loss of one or both eyes is inevitable, as fresh lesions are from time to time appearing in their immediate vicinity.

TREATMENT

Radium is to-day the foremost method of treatment in the eradication of this disease. It is necessary to have the patient continuously under observation, as fresh lesions are constantly appearing, and fresh nodules should be treated immediately they manifest themselves. Excision is a risky procedure in the region of the eye. One of my patients had recurrence, and involvement of the eye after four operations upon the inner canthus. X-rays can be used, but fresh lesions have been attributed to their use, following their application to a primary growth. Carbon dioxide snow can be applied to the early nodules.

PLATES TO ILLUSTRATE MULTIPLE RODENT ULCER
(FIGS. 28 TO 35)

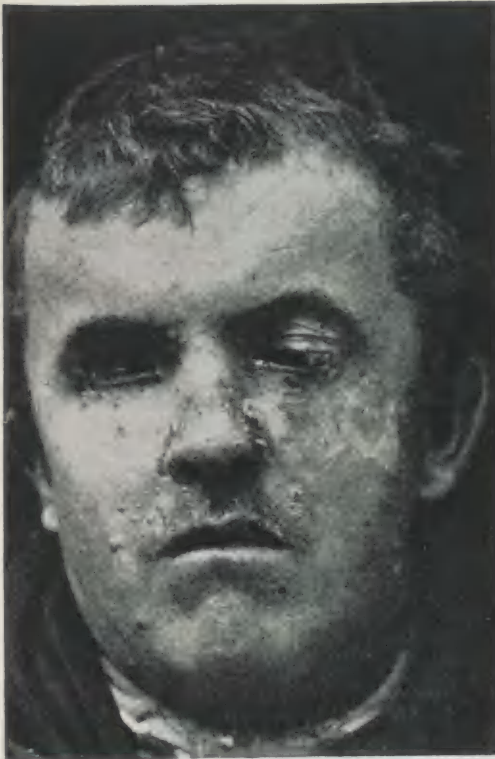


FIG. 28 A and B.—MULTIPLE RODENT ULCER

The patient is the only member of his family to be affected. The disease first manifested itself about six years ago. There are now 30 to 40 nodules varying from pinhead size to 5-6 mm. scattered about the face. They are perhaps more numerous about the eyes, naso-facial folds, and the mouth. The eruption for the most part consists of discrete raised nodules, some of which are pearly in appearance, while others resemble the normal skin in colour. The nodules tend to break down when they reach a certain size, and several of these ulcers are at the present time to be seen on the face. The right eye was enucleated several years ago, on account of one of these growths invading it, whilst the borders of the lids of the right eye present several small growths. The history shows that the scars on the left malar region were caused by radium. The raised growths on the side of the nose are to be attributed to chronic X-ray dermatitis.





FIG 29.—MULTIPLE RODENT ULCER

Æt. 26. Duration three years. Note the numerous small nodules scattered over the face, the superficial type of rodent ulcer just external to the right eye, and the indifferent border of the ulcer on the side of the nose.

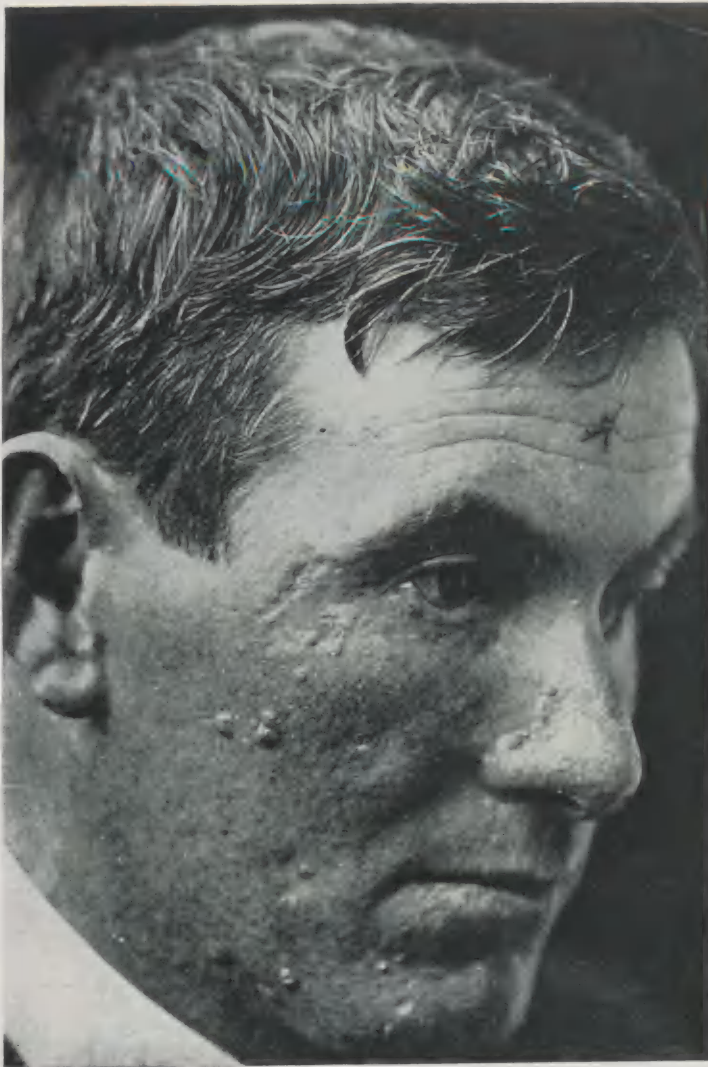


FIG. 30.—MULTIPLE RODENT ULCER

Note the lesion on the side of the nose after treatment by radium. Milia are present on the forehead.

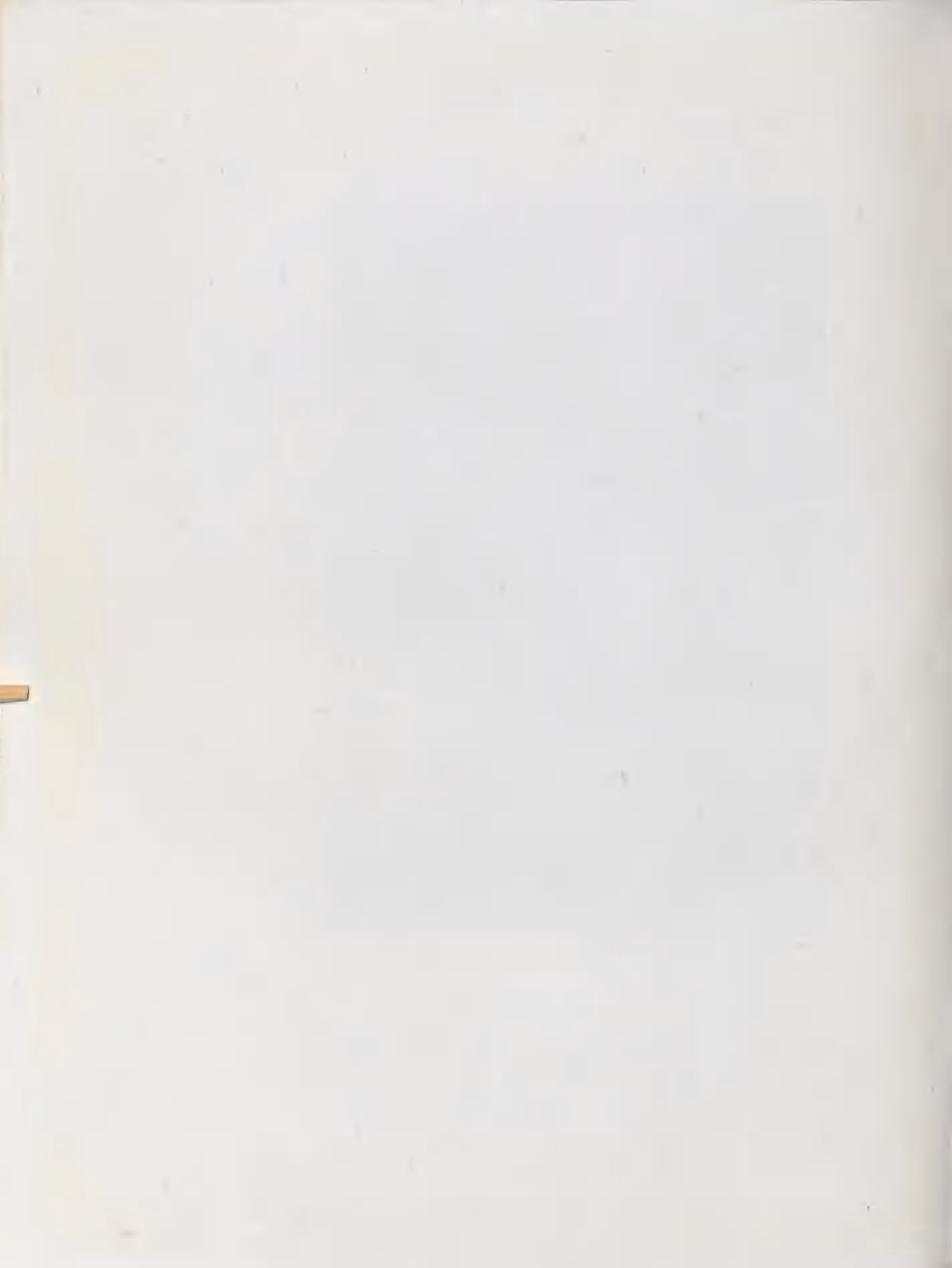




FIG. 31.—MULTIPLE RODENT ULCER

To show right side of the face of Fig. 29. Observe the beaded border of the ulcer at the inner canthus of the left eye, and the absence of a beaded border from the ulcer on the right side of the nose. The right eye is almost destroyed, the patient having had four operations on it before commencing treatment by radium.



FIG. 32.—MULTIPLE RODENT ULCER

Showing lesions on the side of the nose, and inner canthus of left eye after partial treatment by radium.



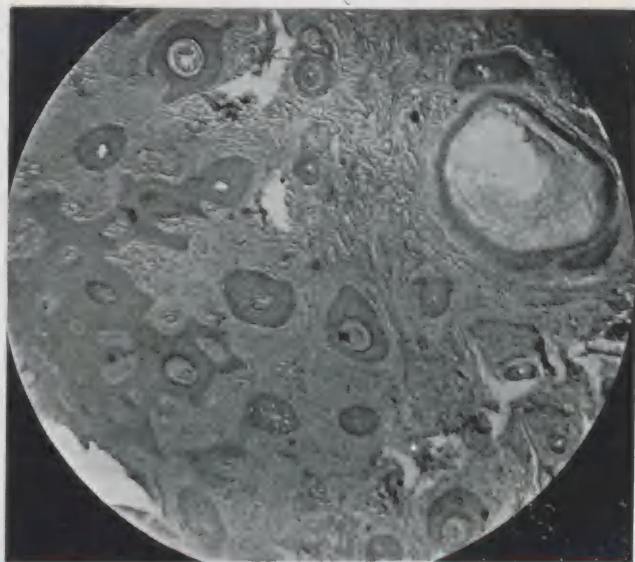
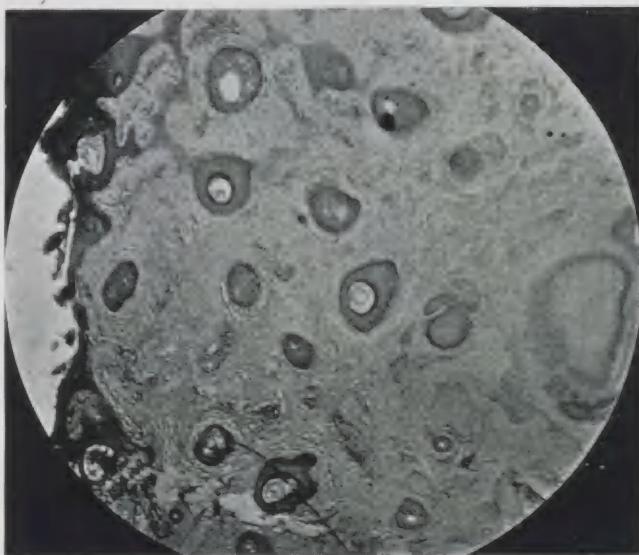


FIG. 33.—MILIUM (Sections 1 and 2)

Section of milium obtained from a case of multiple rodent ulcer (Fig. 30). The cyst is situated somewhat deeply in the corium, independent of and not connected with other structures. Its wall consists of epithelial cells, several layers in thickness. Situated directly above it and near the surface epithelium are outgrowths appearing from the hair follicles. The milium here represented appears to be derived from dilated hair follicles due in all probability to occlusion by the new growth directly superficial to it.



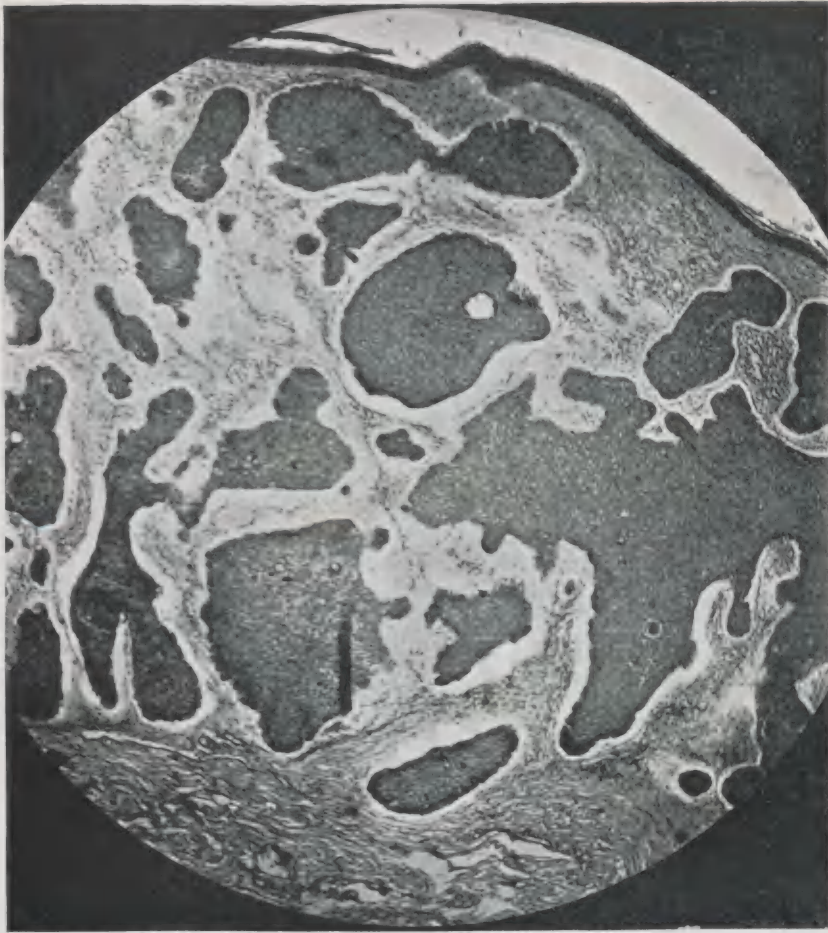


FIG. 34.—MULTIPLE RODENT ULCER

Micro-photograph of section from nodule of the patient seen in Fig. 32. It shows lobulated masses of cells without any evident connection with the surface epithelium. These masses are composed of cells closely packed together and limited by a marginal palisade layer. They are enclosed by somewhat highly organised fibrous connective tissue. Note the absence of any definite small cell infiltration.

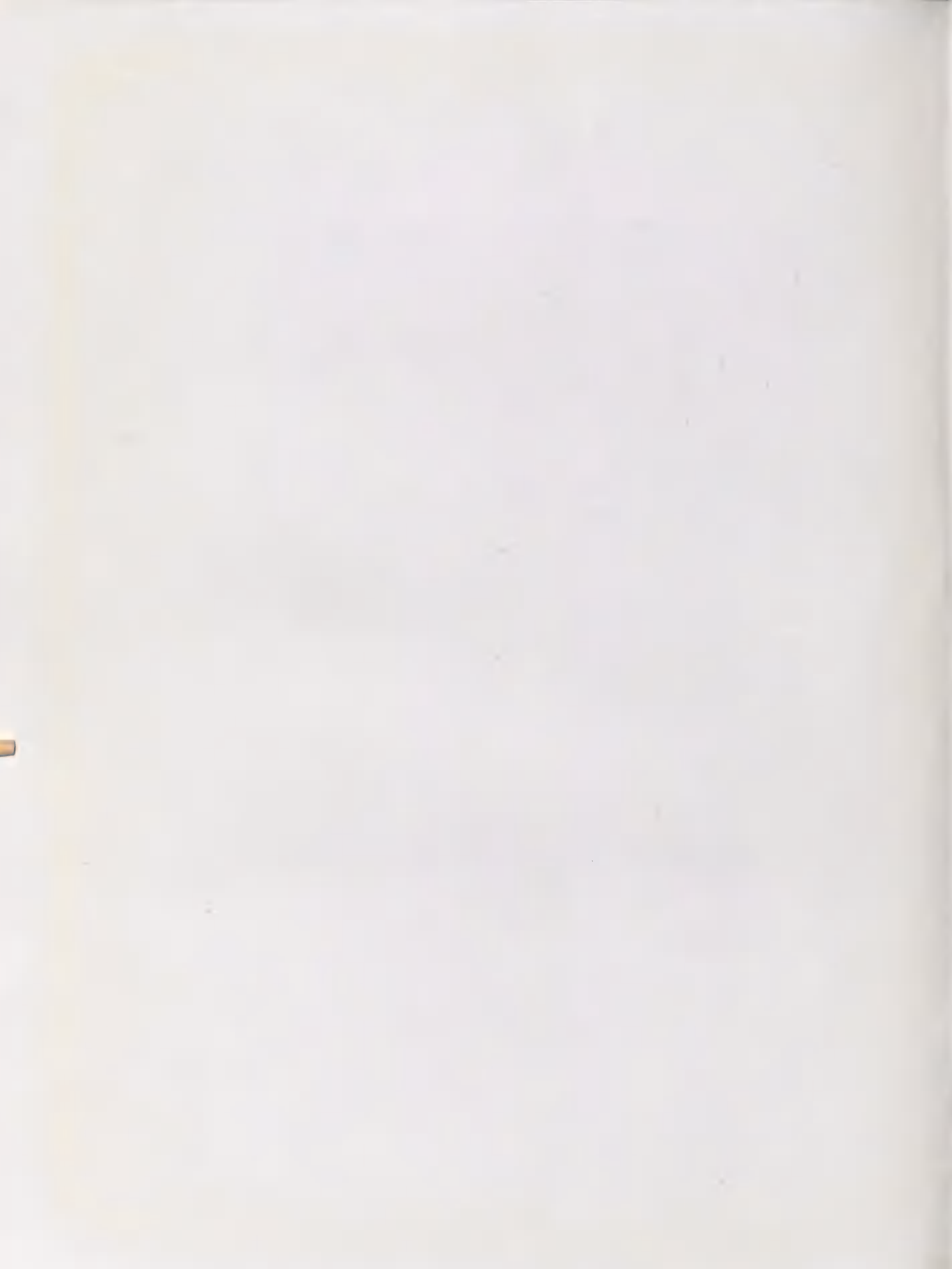




FIG. 35.—EPITHELIOMA ADENOIDES CYSTICUM. (Brooke.)

The father, grandfather, a sister, and two brothers were similarly affected.



TROPICAL SKIN

This is the name which has been applied to a condition characterised by changes analogous to, and identical with, those described in *dermatitis solaris chronica*, but appearing in men in the third and fourth decade of life who have been constantly working in hot climates. Men of fair complexion are most susceptible to these degenerative atrophic changes of the skin. In these patients the pigmented spots or patches are not so pronounced a feature, as they are in the closely allied congenital condition known as *xeroderma pigmentosum*. This dermatosis was named "Sailors' skin" by Unna, because he observed it chiefly in those who spend their life at sea. This is to be explained by the fact that many of these men are constantly traversing tropical regions. Because it is so apt to occur in those who work in the fields, in certain parts of the United States, it has accordingly been termed "Farmers' skin."

PLATES TO ILLUSTRATE TROPICAL SKIN
(FIGS. 36 AND 37)

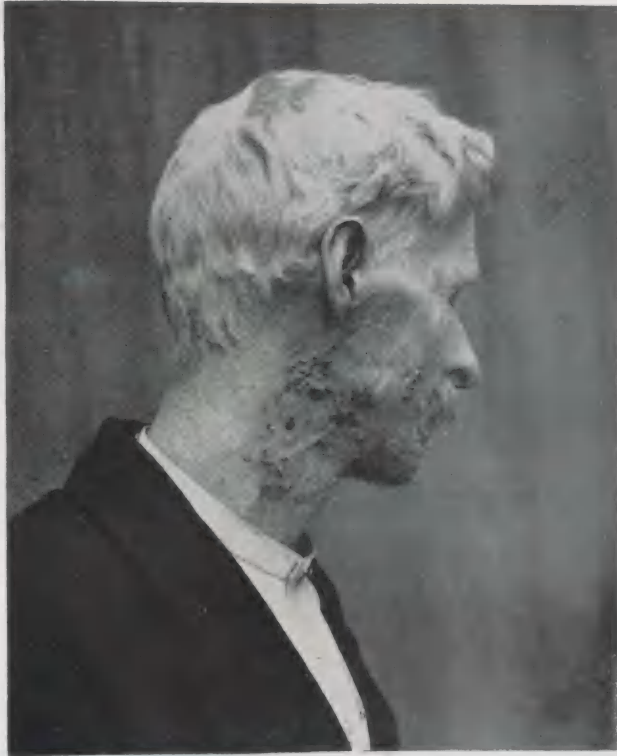


FIG. 36.—TROPICAL SKIN

Duration four years. Multiple keratoses and several small epitheliomata are present on the right side of the face. On the left side of the face there were two small epitheliomata which were excised. Soon after their excision, however, the cervical glands on the same side became enlarged. The patient is a man of fair complexion, much exposed to the weather.

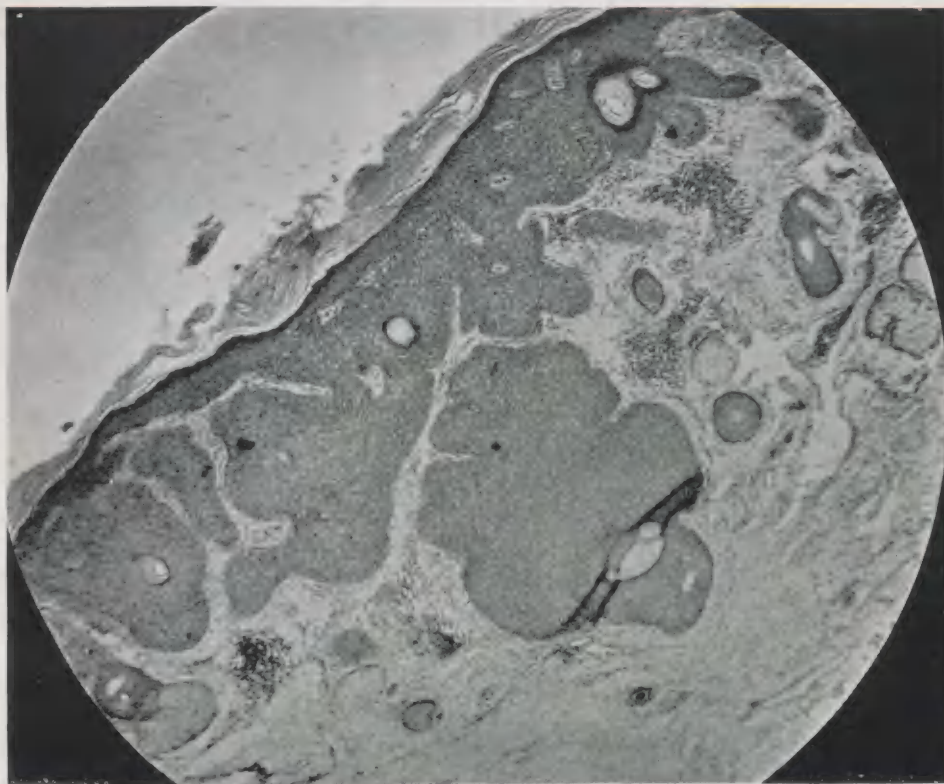


FIG. 37.—TROPICAL SKIN

Micro-photograph of a section of a *keratosis solaris* removed from the face of a patient seen in Fig. 36. The horny layer of the epidermis is slightly increased in thickness. The prickle cell layer is greatly hypertrophied, and for the most part is continuous with the surface epidermis. It is somewhat sharply defined from the surrounding connective tissue, where there is an absence of any marked small cell infiltration, which is usually to be observed when the prickle cells show definite infiltration. The cells composing these down-growths are of the immature prickle cell type, *i.e.*, without prickles and closely packed together. There is no tendency to the formation of cell nests. More advanced growths than the present one were removed from this patient by surgical means, with recurrence in the glands of the neck.

EPITHELIOMA

DEFINITION

Epithelioma is a malignant disease of the skin arising from the epidermis. Its progress is somewhat rapid, and after a variable time involves the neighbouring lymphatic glands, and may produce metastases in the internal organs.

ETIOLOGY

Age is an important factor, as epitheliomata are more prevalent in those of advanced years. The following is a table of the last fifteen cases seen by me :

Sex.	Age.	Site of Distribution.
Female . .	51 . .	Forehead
Female . .	66 . .	Right cheek
Female . .	70 . .	Right hand
Male . . .	72 . .	Ear
Male . . .	58 . .	Neck
Male . . .	69 . .	Right cheek
Male . . .	73 . .	Neck
Female . .	61 . .	Left cheek
Male . . .	45 . .	Right cheek
Male . . .	65 . .	Region of the left ear
Female . .	75 . .	Chin
Female . .	77 . .	Left cheek
Male . . .	68 . .	Hand
Female . .	77 . .	Hand
Female . .	70 . .	Right temporal region

There may be hereditary tendency, as it occurs in childhood in the condition known as *xeroderma pigmentosum*, where the skin shows a strong predisposition to epitheliomata. Significance must be attached to the fact

that the predominating sites are those exposed to strong sunlight, *i.e.* the face, neck, hands, and forearms, and this is undoubtedly one of the chief causative factors in their production. Likewise, long and continued exposure to Röntgen rays may produce this unfortunate result, which was sometimes to be seen following the treatment of *lupus vulgaris*, before the dosage was accurately measured. It may also originate from trauma, scars, cutaneous horns, syphilis, and nævi.

PATHOLOGY

As the growth frequently arises from a keratosis, there may be some hyperkeratosis, and along with this there is downgrowth and widening of the interpapillary processes, so that the prickle-cell layer is increased in thickness. Later, the papillæ become obliterated, and the corium invaded with masses of epithelium, derived from, and continuous with, the prickle-cell layer of the epidermis. The basal-cell layer now no longer forms a limiting margin to the growth, so that the epithelial cells are observed infiltrating the corium. The early histological picture rarely shows cell nests, and the type of cells near the surface may closely resemble those of rodent ulcer in being small and closely packed together, whilst in the deeper portion of the growth the cells may be larger and show prickles. The connection of the underlying growth with the surface epithelium is always well marked and readily discernible, and if the section be removed from the border of the neoplasm the interpapillary processes adjacent will be seen to be hypertrophied. When the proliferating epithelium becomes so extensive as to form large masses, necrosis may take place through failure of the blood supply. Mitoses occur in all parts of the tumour. The

corium shows a marked small cell infiltration beneath the invading masses of epithelium. This seems to vary in inverse ratio to the malignancy of the growth, so that the more benign growths show but a slight infiltration with these cells, whilst the more malignant growths exhibit the corium densely packed with these types of cells. The corium also shows disappearance of the elastic fibres.

SYMPTOMS

Epithelioma may be single or multiple. It most frequently arises from a *keratosis solaris*, so that the initial lesion may be an insignificant scaly spot or warty growth. It may also originate independently as a nodule. These growths increase until they become pea-sized or larger, when they necrose in the centre and break down. The external surface of the nodule slopes gradually from the skin to the summit. The surface shows no burnished appearance, nor are there telangiectases. The internal border of the ulcer may be undermined. Usually at this stage there is no involvement of the lymphatic glands. Later the growth enlarges somewhat rapidly and fungates, and the lymphatic glands may now show enlargement. Epitheliomata are not as malignant as carcinomata arising from a mucous or muco-cutaneous surface. When the growth has attained a large size, the fungating mass becomes purulent, owing to the necrosis, and has a foetid smell. The general signs of malignancy, such as cachexia, wasting, etc., now become evident. Epithelioma may also arise *ab initio* as an ulcer, and this not infrequently takes place beneath a greasy *keratosis solaris*. If this be removed or becomes detached, an ulcer is observed, which may be septic with purulent contents. The border of this ulcer becomes raised, and the growth then advances through the

various stages above described. It is only late in the disease that pain is experienced, which may then become severe.

DIFFERENTIAL DIAGNOSIS

1. *Chancre* may occur on the face, and most frequently is situated in the region of the lip. There is involvement of the adjacent lymphatic glands, and unless syphilis be borne in mind, it may be mistaken for epithelioma. The older the patient, the greater is the likelihood of this error being made. The onset of syphilis is more rapid, and if doubt exists the lesion should be examined for *Spirochæta pallida*.

2. *Gummata* are more rapid in their progress, and at their inception usually involve a larger area and are inflammatory. They break down rapidly, and produce an ulcer with a sloughing base.

3. *Blastomycosis*.—Yeast is attributed to be the cause of this affection, which is characterised by somewhat well-defined, raised lesions, composed of warty growths, often moist with pus, having pin-point openings at various parts of the surface, and especially at the borders. Pus can be made to exude on pressure from these openings.

4. *Rhinophyma*.—This is a hypertrophic form of *acne rosacea* in which there are lobulated outgrowths from the nose, composed for the most part of glandular and connective tissue hypertrophy.

5. *Sebaceous cysts*, when small, may closely resemble epithelioma in appearance, but are more diffuse and not quite so elevated. They are firm and elastic, and fluctuation may be detected.

6. *Fleshy moles*.—These usually make their appearance early in contradistinction to the appearance of epithelioma later in life.

7. *Botriomycosis* is a chronic, benign and usually

pedunculated granuloma, due to infection with pus organisms, and having a raspberry-like appearance.

8. *Dermatitis vegetans* is likewise a benign disease. It is characterised by plaques of exuberant granulation tissue. It arises on some pre-existing lesion. Pus may be free on the surface or present beneath the crusts.

9. *Bromide eruption*.—The lesions in this are multiple. They occur most frequently in children, and there is a history of taking drugs. At first there are hard nodules, which later develop into large granulomatous lesions.

10. *Rodent ulcer*.—It has been stated elsewhere that occasionally the clinical appearances of epithelioma and rodent ulcer may so closely resemble one another as to be indistinguishable, and it will only be possible to make a differential diagnosis by reviewing the more typical lesions of each disease.

DIFFERENTIAL DIAGNOSIS

Rodent ulcer.—Clinically there may occur in both rodent ulcer and epithelioma nodules and ulcers.

A. The nodular stage is characterised by:

Rodent Ulcer.	Epithelioma.
1. The nodule is <i>usually</i> small and pearly in appearance, with dilated vessels coursing over it, but occasionally the nodules reach a larger size.	1. The nodule is always large, and in appearance like that of the normal skin.
2. The growth is dome-shaped, and arises somewhat abruptly from the normal skin.	2. The growth slopes gradually from the summit to the normal skin.
3. In the majority of the cases the nodules break down when they reach split-pea size.	3. Usually reach a larger size than a rodent nodule before breaking down.
4. May take years to reach this stage.	4. Weeks or months only may suffice for the growth to attain the size of a large nodule.

B. Ulcerative stage with an elevated border :

Rodent Ulcer.

1. The border is rolled, beaded or nodular, and slightly raised, but may occasionally be more extensively elevated.
2. Border pearly with vessels coursing over it.
3. Of lengthy duration.
4. Hard and cartilaginous, often giving the sensation of a coin felt between cloth.
5. Glands not involved.
6. The discharge scanty, except when the deep tissues are involved.

Epithelioma.

1. Border is generally more elevated, with a gradual incline to the normal skin.
2. Border fleshy and without vessels.
3. Of short duration.
4. Of softer consistence.
5. Glands involved, usually at a late stage.
6. Discharge more copious.

C. Ulcerative stage without elevated border :

Rodent Ulcer.

1. Usually a late stage and of years' duration.

Epithelioma.

1. Most frequently an early stage, and usually arising as a degenerative process beneath a keratosis. Of short duration.

TREATMENT

Is either by radium or excision.

Radium can be satisfactorily used in all early cases as well as in some moderately advanced cases. Should the growth, however, still enlarge or show no improvement after the first series of treatment, radium should be discontinued. In the treatment of epitheliomata the cosmetic effects must be entirely disregarded, so that excision well beyond and beneath the growth can be undertaken during any period. It must be remembered that a great number of these growths occur in the aged, when operative



FIG. 38.—FUNGATING EPITHELIOMA

Keratosis on the nose.

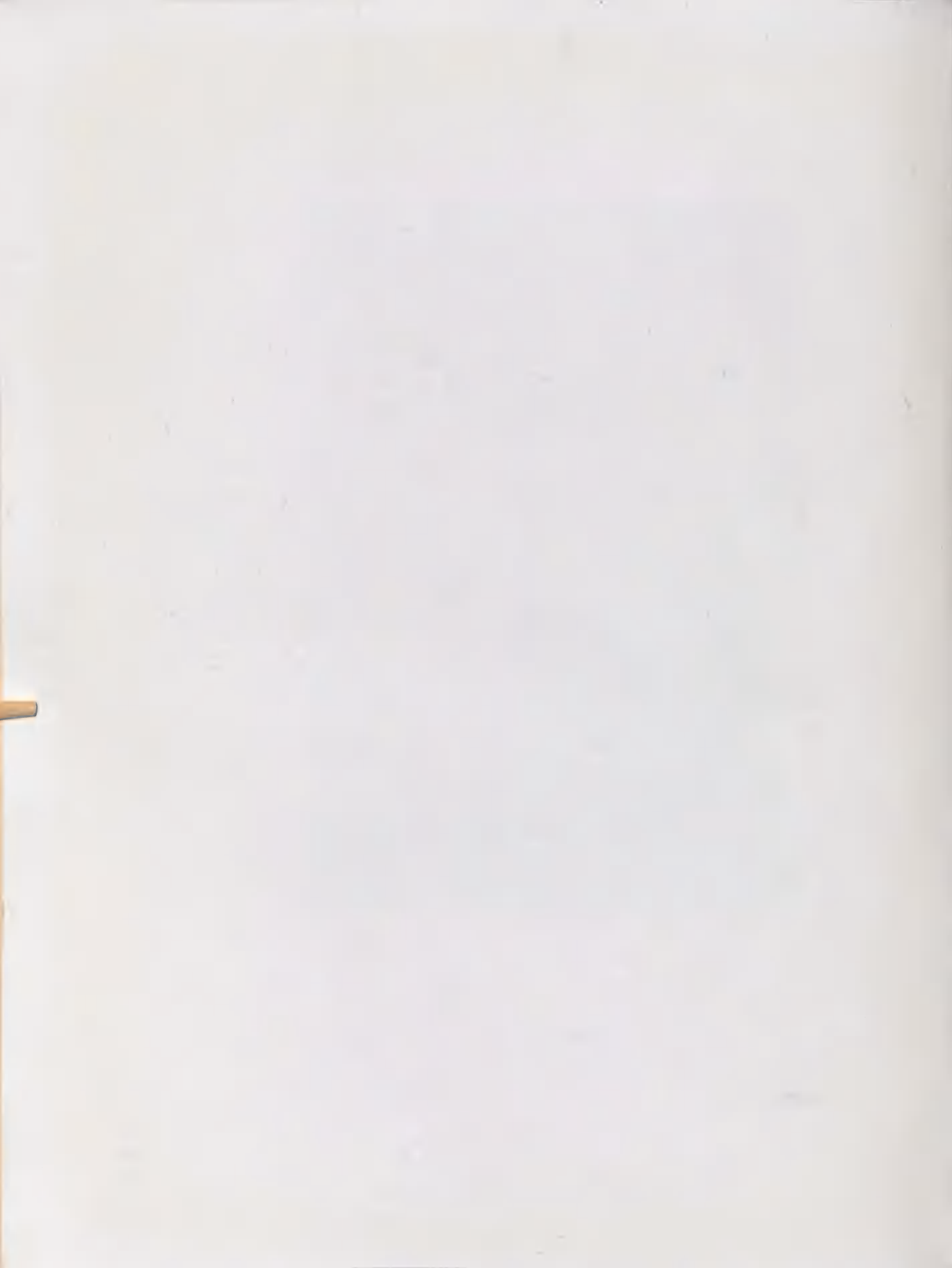




FIG. 39.—EPITHELIOMA OF THE HAND AND RODENT ULCER OF THE FACE

Æt. 77. Discoid epithelioma of the hand, moveable with the skin. Glands absent. It was excised and some time later showed no signs of recurrence, although there was but a small area of tissue removed beyond and beneath it. Rodent ulcer on the tip of the nose with a history of two years' duration. There is also a similar growth on the inner canthus of the left eye. There are also present *keratoses solares* and areas of pigmentation. The patient had a growth removed some years ago from beneath the left eye.



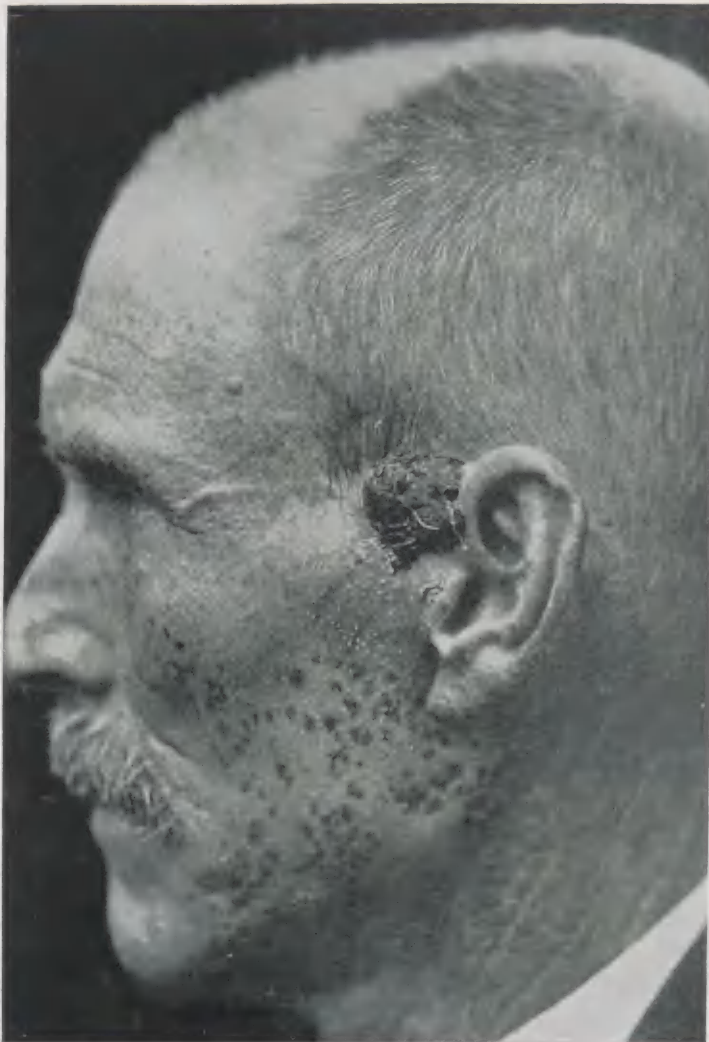


FIG. 40.—EPITHELIOMA

Æt. 65. Duration two years. "Started as a pimple." The dark spots on the face are freckles.





FIG. 41.—EPITHELIOMA

Æt. 45. Duration two months. "Started as a pimple." See also Fig. 42.





FIG. 42.—EPITHELIOMA

After treatment. The resulting scar is due to the destruction of tissue before treatment by radium. Same as in preceding, Fig. 41.





FIG. 43.—EPITHELIOMA

Æt. 73. Duration six months. "Started as a pimple." The growth is raised and vascular in appearance, with a gradual inclined border showing superficial ulceration in the centre.



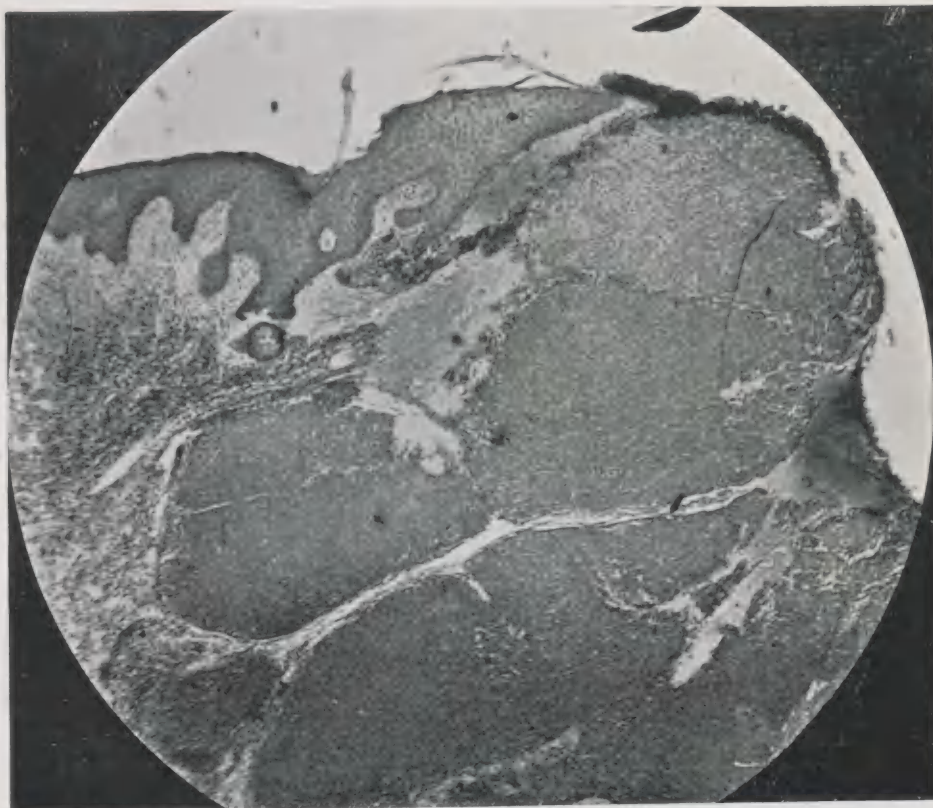


FIG. 44.—EPITHELIOMA

Micro-photograph of a section from the growth on the neck of the patient seen in Fig. 43. There is an extensive downward proliferation arising from and continuous with the epidermis. In the older portions of the growth there is evident differentiation into prickly cells, but in the deeper portions, where rapid proliferation is taking place, the cells are small and closely packed together. They are somewhat sharply limited from the surrounding connective tissue, which shows small cell infiltration. Adjacent to the growth the inter-papillary processes are hypertrophied. Note the absence of cell nests.



procedure is inadvisable, and radium remains the only treatment of choice. Caustics even early should be avoided, whilst ointments only tend to produce increased proliferation of the growth. In incurable epitheliomata pain should be alleviated by the free administration of opium, or morphia.

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